DEPARTMENT OF MEDICINE

DETECTION OF ASYMPTOMATIC URINARY ABNORMA LITIES IN BUNDELKHAND REGION

THESIS FOR

DOCTOR OF MEDICINE
(GENERAL MEDICINE)

BUNDELKHAND UNIVERSITY,
JHANSI (U.P.)

YEAR 2003

ZAHEER HUSAIN

DEDICATED

TO

MY TEACHERS

&

PARENTS

WITHOUT WHOM

THIS STAGE WAS

UNREACHABLE

CERTIFICATE

This is to certify that the work entitled "**DETECTION OF ASYMPTOMATIC URINARY ABNORMALITIES IN BUNDELKHAND REGION**" has been carried out by Dr. ZAHEER HUSAIN in the Department of Medicine, M.L.B. Medical college, Jhansi.

He has put in the required stay in the department of medicine as necessitated by university regulations.

Dated: 7/07/03

Dr. (Prof.) R.C. Arora
MD.,D.Sc.
Professor & Head
Department of Medicine
M.L.B.Medical College,
Jhansi.

CERTIFICATE

This is to certify that the work entitled **"DETECTION OF ASYMPTOMATIC URINARY ABNORMALITIES IN BUNDELKHAND REGION"** which is being submitted as a thesis for M.D. (Medicine) Examination 2003, Bundelkhand University, has been carried out by Dr. ZAHEER HUSAIN under my direct supervision and guidance. The techniques consumed in the preparation of this thesis were under taken by the candidate himself and the observations recorded were checked and verified by me from time to time.

Dated: 07/07/03

Dr. R.K. Jain
M.D., MNAMS
(PROFESSOR)
Department of Medicine
M.L.B. Medical College,
Jhansi.
(GUIDE)

CERTIFICATE

This is to certify that the work entitled **"DETECTION OF ASYMPTOMATIC URINARY ABNORMALITIES IN BUNDELKHAND REGION"** which is being submitted as a thesis for M.D. (Medicine) Examination 2003, Bundelkhand University, has been carried out by Dr. ZAHEER HUSAIN under my direct supervision and guidance. The techniques consumed in the preparation of this thesis were under taken by the candidate himself and the observations recorded were checked and verified by me from time to time.

Dated: 07/07/03

Dr. N.S. Sengar
M.D.,D.M.(Nephrology)
Lecturer
Department of Medicine
M.L.B. Medical College,
(CO-GUIDE)

CERTIFICATE

This is to certify that the work entitled **"DETECTION OF ASYMPTOMATIC URINARY ABNORMALITIES IN BUNDELKHAND REGION"** which is being submitted as a thesis for M.D. (Medicine) Examination 2003, Bundelkhand University, has been carried out by Dr. ZAHEER HUSAIN under my direct supervision and guidance. The techniques consumed in the preparation of this thesis were under taken by the candidate himself and the observations recorded were checked and verified by me from time to time.

Dated:

Dr. N.S. Sengar
M.D.,D.M.(Nephrology)
Lecturer
Department of Medicine
M.L.B. Medical College,
(CO-GUIDE)

ACKNOWLEDGEMENT

It would take pages, to acknowledge everyone who in someway or the other helped me all along my path reaching this goal, but certain individuals need special thanks for their valuable help.

First and foremost ,with all my heart I would like to express my deepest gratitude and privilege for having been associated with Dr. P.K.JAIN M.D. MNAMS, Professor , Department of Medicine, M.L.B. Medical College, Jhansi. I feel extremely fortunate and blessed that I got an opportunity to work under his guidance. His constant teaching, supervision, valuable criticism, affection and encouragement has pruned my work time and again in the right direction and has made this difficult task possible for me. His foot prints shall always be a guidance for me in future life and his achievements shall always encourage and help me find success hereafter.

I wish to express my humble gratitude and regard to my learned and experienced teacher Professor Dr. R.C. Arora M.D. D.sc., Professor and Head Department of Medicine, M.L.B. Medical College, Jhansi . He has acted as a beacon of light to me through his inspiring guidance , valuable and experienced suggestions and indispensable advices at each step in this study.

I am filled with deep sense of obligation towards Dr. N.S.SENGAR M.D.D.M.(Nephrology), Lecturer, Department of Medicine, M.L.B. Medical College, Jhansi. The luminous guidance provided by him in guiding my work time to time has helped me finish this difficult task successfully.

I am also greatly thankful to Dr. PRAVEEN KUMAR JAIN M.D. D.M.(Cardiology) and Dr. NAVNEET AGARWAL M.D. Professors Department of Medicine M.L.B Medical College Jhansi for their constant inspiration and guidance throughout.

I am thankful to my colleagues and juniors who were a delight to work with and my best friend S.Koshy who was a strong support.

Each cell of my body is indebted to my mother Mrs. FARHAT HUSAIN and my father Mr. NAWAB HUSAIN, for their love, affection, care, moral support, blessing and constant encouragement throughout this work . I am also grateful to my brother Mr. SHAHEER HUSAIN and sister Mrs. NIKHAT SULTAN for their constant encouragement.

I owe extreme admiration, love, care and thanks to my sweet and loving wife Mrs. SAIRA HUSAIN for her immense love, devotion, concern and help with which she stood by me throughout the testing times. She shall always be my treasure in life.

I would like to express my thanks to Mr. Shashank Jain ,chief of Netsol Computers for the pain he took in helping me prepare a presentable and neat work to submit.

I shall fail all my thanks if I do not convey all my thanks and respect to all the people who took part in this work and helped it become a complete study which reached conclusion.

Zaheer Husain

DATED: 07/07/03 *(ZAHEER HUSAIN)*

CONTENTS

INTRODUCTION

INTRODUCTION

Near constancy of composition of internal environment of the body, including the volume, tonicity, and compartmental distribution of body fluids and electrolytes is essential to survival.

The kidneys regulate the composition and volume of plasma which in turn determines the composition of volume of intra and extracellular compartments.

Renal diseases may present to physicians in several ways depending upon the nature of the illness and timing of presentation. Some patients present with advanced renal disease, having signs and symptoms of uraemia with unremarkable urinanalysis, while others have urinary abnormalities, but few if any disturbances in renal functions.

SYMPTOMATIC RENAL DISEASE :

Can be of 3 Types :

1. Patients complaining of symptoms or signs which directly or indirectly indicate underlying renal disease.
2. Patients having systemic disease know to be associated with renal involvement.
3. Patients having family history of inherited renal disorders.

Symptomatic renal disease most commonly presents as disorders of micturition, urine volume, urine composition, pain, edema, symptoms of uraemia or symptoms of various disorders involving the kidney secondarily.

(A) DISORDERS OF MICTURITION:

Most common disorder of micturition is frequency. Frequency means that the bladder is emptied more often than normal. Frequency may be associated with increased urine volume (polyuria) or normal urine volume. Frequency with normal urine volume can be due to irritation of the bladder by inflammation, stone, tumor or fibrosis. It can also be due to a pelvic mass or gravid uterus. It is important also to determine whether there is normal or decreased volume per void. The former indicates increased urine formation while the latter indicates diminished bladder capacity. Middle aged and older men with prostate enlargement sometimes present with increased urine volume that results from diminished flow rate in nephrons and impaired concentrating. Ability of the kidney due to obstructive back pressure.

(B) DYSURIA :

Dysuria is pain, discomfort or burning sensation during micturition, it is usually described by the patient as burning or tingling sensation felt at the urethral meatus or in the suprapubic area during or immediately after micturition. It usually arises as a consequence of bladder, prostatic or urethral inflammation. In younger patients it should be suspected if the child cries during micturition or unexplained fever.

(C) DISORDERS OF URINE VOLUME:

Disorders of urine volume can be divided into polyuria (increased volume), oliguria (diminished volume) and anuria (absence of urine).

(a) Polyuria can be due to :

i. excessive compulsive drinking

ii. increase in tubular solute load (urea in CRF, glucose in Diabetes mellitus or low molecular weight proteins in melanoma)

iii. A diminution in ADH production (in head trauma, tumor or infections of hypothalamus or pituitary or sleep disorder)

iv. Disordered medullary concentrating ability as a consequence of medullary disease (Analgesic nephropathy, renal papillary necrosis, medullary cystic disease, sickle cell anemia and nephrocalcinosis).

(b) Oliguria describes a reduction in urine volume to less than that required for the excretion of residues from normal daily metabolic function. In adults under extreme conditions, homeostasis can be maintained with urine output of 500ml per day (1.0 ml / kg / hour in young children). Volume less than this is called oliguria. It usually indicates underlying acute renal failure.

(c) Anuria is absence of renal output and indicates obstruction of urinary tract and rarely renal infarction or cortical necrosis.

(D) DISCOLORATION OF URINE :

Red / Brown discoloration of urine can be due to certain causes like hematuria, haemoglobinuria, Myoglobinuria, porphyrias, urates, alkaptonuria, drugs and dyes. Blood arising from the glomerulus gives urine a smoky appearance or tea / coca cola appearance. Blood arising from the urethra usually appear at the beginning of urinary stream while that from the bladder or prostate is commonly noticed at the end of micturition.

(E) PAIN :

Pain is an inconsistent symptom of urinary disease but when present is commonly due to obstruction or inflammation. pyelonephritis usually causes pain at renal angle. A perirenal abscess can give symptoms related to diaphragmatic irritation. Glomerular inflammation can be associated with dull lumbar ache. Pain arising from an acute obstruction is usually sudden in onset, severe colicky in nature and radiates from loin to groin.

(F) Oedema :

May arise due to hypoproteinemia which is a consequence of significant proteinuria. The edema is usually most noticed around the eyes in the morning and in the ankles and feet in the evening. Edema may also arise as a consequence of salt and water retention in cases of acute or chronic renal failure.

(G) CLINICAL ABNORMALITIES IN URAEMIA :

Uremia refers to the retention of nitrogenous wastes as renal insufficiency develops and causes multiorgan system derangements which become clinically manifest.

a. Fluid electrolyte and acid base disorder -

Can develop and lead to volume expansion/contraction; hypo/ hypernatremia; hypo/hyperkalemia; metabolic acidosis; hypocalcaemia and hyper phosphate.

b. Endocrine metabolic disturbances -

Include secondary hyperparathyroidism; vit-D deficiency; Carbohydrate intolerance; hypertriglyceridemia; protein calorie malnutrition; impaired growth; infertility; amenorrhea & sexual dysfunction.

c. Neuromuscular disturbances-

Caused due to uremia are fatigue; sleep disorder, headache, impaired mentation, asthenias, seizures, cramps, peripheral neuropathy, myopathy & comes.

d. Cardiovascular & pulmonary disturbance-

arterial hypertension; CHF; pulmonary edema; pericarditis; hypertension and arrhythmia.

e. Dermatologic disturbances-

Pruritis ; ecchymosed ; hyperpigmentation.

f. Gastrointestinal disturbances-

Include anorexia; nausea ; vomiting ; Gastroenteritis ; peptic ulcer ; G.I. bleeding ; peritonitis ; hepatitis.

g. Hematologic & immunologic disturbances-

Includes anemia; lymphocytopenia ; bleeding diathesis; infection; splenomegaly.

ASYMPTOMATIC RENAL DISEASE

Is most commonly detected following routine investigations such as urine analysis, blood pressure or blood chemistry analysis after hospitalization for non renal causes, or as part of health screening programs. In a number of patients renal disease is detected during clinical and laboratory test for pregnancy, occupational purposes or health insurance. A considerable number of neonates are diagnosed as having renal disorders because of routine USG screening of mothers during pregnancy. In a small number of cases there is regular screening in view of a known employment and development of renal disease (aniline due workers have a greater incidence of urothelial tumors)

PERSISTING URINARY ABNORMALITIES WITH NO OR FEW SYMPTOMS.

The finding of hematuria , proteinuria, bacteriuria , crystalluria and Pyuria in absence of readily identifiable disease are familiar and vexing clinical problems .

(A) ASYMPTOMATIC HEMATURIA:

Determining the morphology of erythrocytes in freshly voided urine is useful to separate glomerular form nonglomerular hematuria (fragmented distorted poorly hemoglobinized or dysmorphic RBC with a volume of less than 72 fl are usually glomerular). Glomerular hematuria is frequently accompanied by proteinuria (non nephritic range). Patients with isolated hematuria with minimal urinary symptoms are likely to have no or minimal glomerular changes on electron microcopy and are usually caused by SLE, Wegner's granulomatosis ,Goodpasteur's syndrome , Alport's syndrome , idiopathic hypercalcemia , thin basement membrane , silent Nephrolithiasis or malignancies.

(B) ASYMPTOMATIC PROTEINURIA:

Is defined by the pressure of mild glomerular proteinuria (principally albumin) usually less than 2 gm/day with normal urinary sediments in the absence of symptomatic systemic disease. Asymptomatic essential hypertension and mild or latent Diabetic Nephropathy, Idiopathic membranous glomerulonepheritis, Focal glomruloscleresis, IgA nephropathy, and Amyloidosis may all present initially with mild or moderate proteinuria. Other uncommon causes are Postural proteinuria, Over flow proteinuria and Tubular proteinuria.

(C) ASYMPTOMATIC PYURIA:

Upto 400,000 cells / hour may be excreted in normal urine, corresponding to 10 WBCs / ml in an unspun urine sample. Many times present of pus cells in the urine can be detected in the absence of clinical symptoms. This is known as asymptomatic pyuria . Common causes of asymptomatic pyuria are infection , Diabetes mellitus, NSAID Nephropathy , Renal tuberculosis , Interstitial nephritis , Nephrolithiasis and prostatic enlargement in males.

(D) ASYMPTOMATIC GLYCOSURIA:

Presence of even small amounts of glucose in the urine sample is an abnormality. Asymptomatic glycosuria in individuals is almost always due to diabetes mellitus which has not clinically manifested or detected.

(E) ASYMPTOMATIC CRYSTALLURIA:

Except for the cystine crystals and a few others, the majority of crystals found in the urinary sediment are of limited value. It is tempting to associate crystals with a risk of Nephrolithiasis. In a majority of cases, the crystals found in the urine are not present in freshly voided sample . some of the common causes of asymptomatic crystalluria are urolithiasis , infection , primary hyperparathyroidism , excessive bone resorbtion , ethylene glycol toxicity , renal tubular acidosis , chronic diarrhoea and drugs .

AIMS & OBJECTIVES

AIMS AND OBJECTIVE

1. To detect the prevalence of asymptomatic urinary abnormalities in Bundelkhand region and to find out the prevalence of diseases that were causing these urinary abnormalities to appear.

2. To compare the results of this study with other studies done in other parts of the country and in different regions of the world. This gave an idea of the changes in prevalence and causes of asymptomatic urinary abnormalities in different age groups in different regions in our country and outside.

3. To show the importance of urinalalysis as a valuable and efficient method of early detection and intervention of urinary abnormalities.

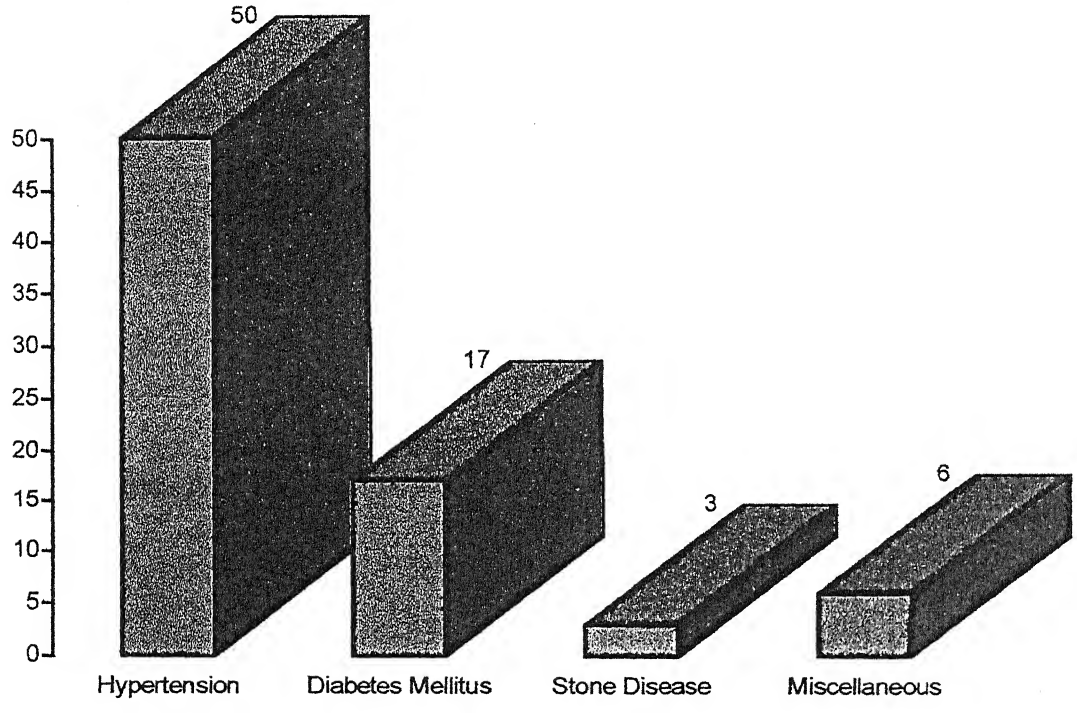
REVIEW OF LITERATURE

REVIEW OF LITERATURE

A number of studies have been conducted using urine examination as the tool for detection of asymptomatic urinary abnormalities and surprisingly vast majority of causes have been found out all differing between age, sex, socio economical status and geographic distribution.

Kidney disease detection camp (1994-1996) - A tool for preventive Nephrology conducted by – Vidya N. Acharya, Kumad Mehta et al This study involved 430 subjects seen in 3 Kidney disease detection camps held in the city of Mumbai to cover low socio economical group (LSEG.,nz 244) and mid socio economical group (MSEG;nz 186). The aim was to detect the presence of urinary abnormalities and assess the significance of the same. Subjects attending the camp had their vital data and BP recorded in a specially designated Performa. A fresh sample of urine was obtained from each one of them and examined by using multistix. Those having any clinical or urinary abnormity were reassessed next morning & findings were confirmed. Urinary abnormalities were detected in 42.1% of total subjects (181/430) of which 45.5% were in low-socioeconomic group & 37.6% in mid-socio economic group. In this total of 181 urinary abnormalities 37.8% (111/244) were asymptomatic from LSEG and 47% (70/156) from MSEG. The study found that Asymptomatic urinary abnormalities were associated with hypertension (19.8% in LSEG & 40% in MSEG-P<0.001); Diabetes mellitus (8.1% LSEG & 11.4% in MSEG) and stone disease (1.8%in LSEG & 1.4MSEG).

Kidney Disease Detection Camp(1994-1996) TYpe of Disease Detected in LSG + MSEG(181)



In advanced countries like Japan mass screening programmes with compulsory urine screening in children enabled them to detect disease in pre-symptomatic stage successfully. Such studies have been done in adult population too in Japan (all since 1947). During these last 40-50 years, they have been also to detect pre-symptomatic glomerulopathies of various types like IgA nephropathy, membrane proliterative GN etc. and have recommended early treatment for the same. This study her revealed that main causes of urinary abnormalities in our Community has been in association with common maladies like Hypertension (27.6%); Diabetes mellitus (9.4%) which affect the population at large. Simple urine examination on yearly basis would certainly help in early detection of renal involvement and thereby form a valuable tool in the practice of preventive Nephrology.

To define the long-term outcome of patients with minimal urinary abnormalities (defined by the presence of microscopic hematuria with on less than 1 gm/day proteinuria), and normal renal function (defined by a serum creatinine< 1.3 mg/dl), **Shu KH, Ho WL, Lu YS, Cheng CH, Wu MJ, Lian JD Department of Internal Medicine,Taichung Veterans General Hospital, Taiwan** retrospectively studied patients who fulfilled the above criteria and had a kidney biopsy done before the year of 1992 (i.e. at least followed up for 5 years), with a definite pathological diagnosis. A total of 41 cases among 719 cases of primary glomerulonephritis (5.7%) were enrolled into the study. There were 19 males and 22 females with a mean age of 35.4+/-14.7 years at biopsy. The duration of renal disease was 116.0+/-60.5 months and the duration of follow- up post biopsy was 100.2+/-38.1 months. The pathological diagnosis was : IgA nephropathy (21 cases) , focal glomerulosclerosis (9cases), mesangial proliferative glomerulonephritis (8cases) , membranous glomerulonephritis (2cases) and acute glomerulonephritis (1cases) . At the end of follow-up, 8 cases (19.5%) had a certain degree of renal insufficiency including 2(4.9%) in end- stage renal disease. The other cases were either in complete remission (6cases) or stable condition (27cases) with persistent microscopic hematuria and normal renal function. The long-term outcome was not correlated with any of the following parameters: age, sex , disease duration , serum creatinine at presentation, daily protein loss at presentation , degree of glomerular change and degree of tubular atrophy ($P<0.05$) and interstitial fibrosis($P<0.05$).They conclude that a minimal urinary abnormality with normal renal function at presentation does not necessarily imply a favorable long-term outcome in certain patients. Tubular atrophy and interstitial fibrosis but not glomerular change correlates with a worse prognosis. This further emphasizes the importance of renal biopsy in such cases.

Monhart V, Marek J, Krnch O Ustredni vojenska nemocnice, Praha studied 103 subjects with asymptomatic isolated haematuria (Persisting for more than 6 months in the absence of proteinuria, bacteriuria, impaired haemocoagulation or urological disease) renal biopsy was preformed. The mean age of the patients was 25.2 years, range 14-58 years. In 94% glomerular changes were detected- most frequently minimal glomerular lesions (67%) and proliferative mesangial glomerulonephritis (15%) . Focal segmental proliferative glomerulonephritis was rare (4%). Immunofluorescent examination revealed IgA nephropathy in 40% (all cases of diffuse and focally segmental proliferative glomerulonephritis and one quarter of minimal glomerular lesions). Changes of tubules and interstitium were recorded in 26%, with the exception of one patient they were always associated with glomerular affection. From the investigation ensues that the predominating cause of isolated asymptomatic haematuria, not clarified by non-invasive examination, is usually not serious and is an affection frequently associated with tubulointerstitial changes. As many as 40% of isolated cases of haematuria may be the manifestation of IgA nephropathy. The deposition of IgA is more frequently associated with a more advanced grade of glomerular affection; Indication of diagnostic renal biopsy in isolated haematuria remains individual.

. **Topham PS, Harper SJ, Furness PN, Harris KP, Walls J, Feehally J Department of Nephrology, Leicester General Hospital , UK,** investigated 165 patients (94 male, 71 female; mean age 37.5 years, range 10-71) referred with isolated microscopic hematuria with normal serum creatinine, no proteinuria, sterile urine and a normal IVU. Renal biopsy abnormalities were found in 77/165 (46.6%). IgA nephropathy (49), global or segmental mesangial proliferative glomerulonephritis without IgA deposits (16) , thin membrane nephropathy (7), vascular changes

suggestive of hypertension (3), interstitial nephritis (1), and membrane nephropathy (1). Only five abnormalities were found on cystourethroscopy (cystitis3, urethral stricture1, and bladder stone 1). Two patients with cystitis also IgA nephropathy. Biopsy abnormalities were commonest under the age of 20(69.2%) , but 40% of biopsies were abnormal even in the seventh decade of life, Because renal biopsy abnormalities are very frequent in patients with isolated haematuria , renal biopsy is indicated in patients over 45 years of age if renal imaging and cystoscopy are normal . In those under 45 years, renal biopsy should replace cystoscopy as the investigation to follow normal renal imaging.

Hrvacevic R, Dirnitrijevic J, SpasicP, Butorajac J, Jovanovic D analyzed histopathologic changes in the kidneys of patients with asymptomatic abnormalities of urine analysis if they were correlated with the type of pathologic finding in urine. Retrospective study comprised a total of 76 patients with asymptomatic urine abnormalities. In all three groups of patients, formed upon the type of pathologic finding in urine, were determined heterogeneous histopathologic changes, and different types of glomerulonephritis, respectively. The most frequent histopathologic finding was IgA nephropathy, observed in 16.7% patients with isolated proteinuria, in 50% patients with isolated microscopic hematuria and in 55.9% patients with associated urine abnormalities, In distinction from the other two groups of patients , in the group of patients with isolated proteinuria normal histologic finding was very frequently found (25% patients,) , and in group of patients with asscioted urine abnormalities were observed more severe hestopathologic forms of glomerulonephritis, such as membranoproliferative Glomerulonephritis. It was concluded that different types of glomerulonephritis most frequently caused asymptomatic abnormalities of urine in younger patients. In a

prospective study of idiopathic glomerulonephritis ,**Nieuwhof C, Doorenbs C, Grave W, de Heer F, de Leeuw P, Zeppenfeldt E, van Breda Vriesman PJ, Department of Immunology, University of Limburg ,Maastrict, The Netherlands,** determined the natural history of 49 adult patients (12 primary IgA nephropathy, 13 thin GBM nephropathy, 20 normal renal tissue and 4 miscellaneous nephropathies) who presented with idiopathic non-proteinric non-azotemic hematuria of at least six months duration in the absence of hypertension and with a negative urological work- up . The median follow-up was 11 years with a range of 8 to 14 years. At the end of the follow-up, renal function had remained stable in all subsets except for those with miscellaneous disease. Hematuria was still present in all patients with thin GBM nephropathy, in all but two patients with IgA nephropathy who went into immunopathological remission, in three out of four miscellaneous nephropathies, and in seven out of 20 patients with normal renal tissue. Of the latter patients five had a history suggestive of urolithiasis at follow-up, which was in the absence of hypercalciuria and hyperuricosuria. Seven thin GBM patients, five IgA nephropathy patients and three miscellaneous nephropathies developed hypertension; the incidence of hypertension in each subset was significantly higher than in patients with normal renal tissue. This study shows that in young adults with idiopathic chronic non-proteinuric hematuria of four years duration, renal biopsy will give a definite diagnosis in 86% patients , and that those patients with so- called minor glomerular disease are at high risk for hypertension. Those patients with normal renal tissue have a high incidence of urolithiasis and should have a urological follow-up,

In a mass screening programme- **Hisano S, kwano M, Hatae K, Kaku Y, Yamane I, Ueda K, Uragoh K, Honda S Department of**

pediatrics, Faculty of Medicine, Kyushu University , Fukuoka, Japan-- screened 251 children with isolated microhaematuria . Of these 251 children, 115 were excluded from the study because of microhaematuria secondary to a specific cause. The remaining 136 children were diagnosed as having asymptomatic isolated microhaematuria (ASH). Of these 136 children, 23 had evidence of urinary abnormalities in their family members, Red blood cell casts were evident in 31 children at their initial visit or during the follow-up period. Ten children had one or more episodes of macrohaematuria during the study. Renal biopsy was performed in 19 children because of indications of glomerular disease, and 13 of these 19 children had mild to moderate glomerulonephritis. None of these 136 children developed hypertension or renal impairment after a mean period of 7.4 years (range6-13 years), Thirty –five children had normal urinary findings within 6 years of their initial visit , and 100 have had persistent microhaematuria with proteinuria greater than 1g/ m_2 per day at the end of the study . This study suggests that the prognosis of ASH is good, and that renal biopsy is not indicated for children with ASH.

Benbassat J, Gergawi M, Offringa M, Drukker A Department of Sociology of Health, Faculty of Health Sciences, Ben- Gurion University , Beer- Sheva , Israel-- reviewed published data on the frequency of underlying disorders in schoolchildren with microscopic or gross isolated haematuria (IH), and evaluated management strategies. They found five reports of microscopic IH in screened asymptomatic schoolchildren, three reports of microscopic IH detected by case-finding, and five surveys of kidney biopsies in referred children with microscopic and gross IH. They listed the reported underlying disorders, and estimated the benefit from their early detecting and treatment. Most children with microscopic IH, whether detected by screening or case-finding, had no

significant underlying disease. Some had disorders that may benefit from early treatment (membranoproliferative glomerulonephritis, obstructive uropathy, urolithiasis), or counselling (hereditary nephropathy, renal cystic disease). The combined prevalence of these five diseases was 0-7.2% in children with microscopic IH detected by screening, and 3.3%- 13.6% in those with microscopic IH detected by case – finding. The combined prevalence of membranoproliferative glomerunephritis and hereditary nephropathy among kidney biopsies was 11.6% -31.6% in children with microscopic IH, and 3.6% -42.1% in children with gross IH, Variable management strategies for schoolchildren with IH result from uncertainly about the frequency of underlying disorders and the efficacy of their early treatment , With no evidence that detecting III leads to prevalence of renal function impairment, screening for IH in symptom less schoolchildren is not warranted. Once detected, however, IH justifies further investigation.

Since 1998, mass urine screening tests have been performed on Korean school children. **Cho BS, Kim SD, Choi YM, Kang HH Department of Pediatrics, College of Medicine, Kyung-Hee University Hospital, 1 Hoegi-dong Dongdaemun-ku, Seoul, Korea** have analyzed those patients who showed abnormal urinary findings in the school screening program. Between January 1998 and January 2000, 452 children with abnormal urinary findings visited the Pediatric Kidney Center, Kyung-Hee University Hospital. Sex, age, 24-h urine creatinine clearance, ultrasonography, Doppler scans and renal biopsies were reviewed retrospectively. Results of initial urinalysis were divided into three groups: solely hematuria group (228 cases, 50.4%), solely proteinuria group (98 cases, 21.7%), and combined hematuria and proteinuria group (79 cases, 17.5%). Among the biopsies cases, the proportions representing renal

parenchyma diseases were as follows: IgA nephropathy 11.3%, mesangial proliferative glomerulonephritis 21.9%, others 3.8%. Among the three groups, the combined hematuria and proteinuria group had more frequent chronic renal disease (57.7%) than the other groups. Chronic renal disease was detected in 36.9% of all visiting subjects. In the school screening program a significant number of patients showed abnormal urinary findings, which were associated with chronic renal diseases especially in the combined hematuria and proteinuria group. In conclusion, mass urine screening tests should be mandatory to detect asymptomatic chronic renal disease in school children.

Lin CY, Sheng CC, Lin CC, Chen CH, Chou P. Department of Pediatrics, Section of Immunology & Nephrology, Taipei Veterans General Hospital, No. 201, Sec. 2, Shih-Pai Road, Shih-Pai, Taipei, 11217, Taiwan screened students of public and private elementary and junior high schools in the Province of Taiwan each semester since 1990. About 3 million students were screened each time. The students who had abnormal urine screening results at the first time received a second urine analysis 10 to 15 days later to confirm the abnormal urine analysis. The blood samples of the students with abnormal urine examination were taken and biochemistry examinations including creatinine (Cr) etc. were performed since 1992. All students with abnormal urine screening results were graded by the severity of hematuria and proteinuria, the heavy proteinuria graded as "D". Chronic renal failure (CRF) is defined as impaired renal function with the serum Cr over 1.7 mg/dl. Longitudinal continuous blood and urine examinations were performed each semester for the students of grade "D" and with CRF. CRF was confirmed by either the hospital medical records or telephone visit. The purpose of this study was to delineate the prevalence of heavy proteinuria (grade D) and CRF in

the students of elementary and junior high school in the Taiwan Province from 1992 to 1996. The results revealed the number of urinary screening was 10,288,620. There were 5980 cases with heavy proteinuria with four-year prevalence of 5.81 x 10(-4), 4.83 x 10(-4) for boys; 6.87 x 10(-4) for girls. Girls were affected more often than boys. The peak age of girls was 12 years old and boys were 13 years old. The number of CRF cases was 189 with the four-year prevalence of 1.84 x 10(-5), 2.24 x 10(-5) for boys; 1.41 x 10(-5) for girls. The incidence rate increased after the age of 10; the peak age of boys being 14-year-old and of girls 12-year-old. The exact contributing factors, such as location on islet or lack of pediatric nephrologist, need further study. In conclusion, the four-year prevalence of heavy proteinuria in the students of the elementary and junior high schools in Taiwan was higher in girls than in boys. Glomerular nephritis (GN) is still one of the major causes of urinary abnormalities. The most-important secondary GN was systemic lupus erythematosus (SLE) with lupus nephritis. The percentage of SLE patients among anti-nuclear antibody (ANA) positive was 72%. In contrast, the four-year prevalence of CRF disease was higher in boys with the peak age at 14-year-old. GN is still the major cause of urinary screening abnormality. ANA study is indicated in all Chinese students with abnormal urinary screening.

Clinicohistopathologically, **Takebayashi S, Yanase K. Second Department of Pathology, School of Medicine, Fukuoka University, Japan** observed 109 patients with asymptomatic urinary abnormalities found via the Japanese school medical screening process. Follow-up was for a mean period of 9.3 +/- 4.0 years. More than 80% of the patients had either IgA nephropathy (IgAN, 47 cases, 43.1%), thin membrane disease (TMD; 21 cases, 19.3%) or normal glomerulus (NG; 20 cases, 18.3%). Complete remission appeared in 60.0% of the NG cases, 14.3% of the

TMD cases and in 19.1% of the IgAN cases, and remission was significantly high in the NG group (p less than 0.01). No patient with TMD and NG ever progressed to the extent of pronounced proteinuria or renal failure. One patient deteriorated and required hemodialysis, and 2 patients developed renal insufficiency in IgAN. All of these cases possessed severe glomerular sclerotic change when the initial biopsies were performed. All IgAN cases that went into remission, however, had minor glomerular abnormalities. A positive family history of urinary abnormality was observed in 14.1% of both the IgAN group and the NG group, whereas we observed 71.4% in the TMD group, which was significantly high (p less than 0.01). Other cases included 4 each with non-IgA proliferative glomerulonephritis, focal segmental glomerular sclerosis, membranoproliferative glomerulonephritis and Alport's nephritis.It was concluded that the majority of patients (80.7%) with urinary abnormalities found via the school screening program had IgAN, NG or TMD. 74.5% of the IgAN group and 85.7% of the TMD group had long histories of urinary abnormalities extending into adulthood with no deterioration of the renal function.

Wei JN, Chuang LM, Lin CC, Chiang CC, Lin RS, Sung FC. Institute of Environmental Health, National Taiwan University College of Public Health, 1 Jen Ai Road, Section 1, 100, Taipei, Taiwan (1993-1999), did a mass screening programme to describe the gender differences in cases and characteristics of diabetes mellitus (DM) that can be identified from a mass urine screen program for school children in Taiwan.Screening for the childhood asymptomatic proteinuria and glucosuria began in 1992 for school children. Students were instructed to collect mid-stream samples of the first morning urine for glucosuria and proteinuria tests using urine strip devices. Students with positive results for

glucose and/or protein and/or occult blood in the first examination received a second urine test. The third screening test was performed for urine and fasting blood sample for 11-item examinations if the second test was positive. The 1997 criteria of American Diabetes Association were used for defining DM. Approximately 2615000-2932000 students received the preliminary screening each semester. The overall average rates of newly identified diabetes from 1993 to 1999 were 8.3 per 100000 among boys, and 12.0 per 100000 among girls. The average rate of new cases increased significantly from sixth grade for boys and fourth grade for girls, with peak rates of 14.7 per 100000 in eighth grade for boys and 19.0 per 100000 in sixth grades for girls. Similar prevalence trends by sex and grade were observed, higher in girls than in boys. This mass screening data suggest that childhood diabetes of all types in Taiwan is elevated in the age of puberty and higher in girls than in boys.

By governmental mandate, Japanese school children are screened annually for proteinuria, hematuria, and glucosuria to identify children with possible renal disorders. **Pugia MJ, Murakami M, Lott JA, Ohta Y, Kitagawa T, Yamauchi K, Suhara Y, Kasjima J.** added urine dipstick tests for albumin and creatinine to the Japanese screening protocol, and used their dipstick results for blood, glucose and protein. The sulfosalicylic acid precipitation test was used to confirm "trace" positive protein dipsticks. The Japanese and our screening protocol have in common the same data for glucosuria and proteinuria. Their scheme has an algorithm for repeat testing of children with abnormal results, and further testing and medical evaluation for those showing persistently abnormal values. Out of the 23,121 students, we found seven with likely nephritis, one with confirmed nephritis, one with nephrotic syndrome, 170 with persistent unexplained hematuria, 19 with persistent unexplained proteinuria, 14 cases of urinary

tract infection, and 20 cases of likely diabetes mellitus. We conclude that dipstick testing for albumin, protein, creatinine, glucose and occult blood has significant value in a multilevel testing scheme for identifying children with urinary tract abnormalities or diabetes. The assay of albumin increases the sensitivity of the screening, and dividing the albumin by the creatinine concentration reduces the potential errors arising from concentrated or dilute urines.

Beginning in 1974, the Japanese Ministry of Health Welfare directed the screening of schoolchildren for proteinuria. **Pugia MJ, Lott JA, Kajima J, Saambe T, Sasaki M, Kuromoto K, Nakamura R, Fusegawa H, Ohta Y** studied their procedure and methods in 6197 school children and also evaluated a new urine dipstick that measures albumin concentrations down to about 10 mg/l and creatinine down to about 300 mg/l. They used specimens from adult in- and outpatients to test the accuracy of the dipsticks. Based on the quantitative results, they set as cutoffs < 150 mg/l for protein and < 30 mg/l for albumin as the concentrations representing "low risk." The quantitative values were assumed to be correct, and the dipstick results were judged accordingly, i.e., a dipstick protein of > or = "150" mg/l or an albumin of l "30" mg/l indicated increased risk of developing or having a genitourinary disorder. The sensitivity/specificity of the protein dipstick was 95.1%/95.5%, and the same for the albumin dipstick was 83.8%/93.8%. The cut-off for the albumin dipsticks probably should be set somewhat lower to reduce the number of false negatives and increase the sensitivity of the dipstick. When they compared the quantitative albumin to the protein dipsticks with the above cut-offs, they found the sensitivity/specificity to be 79.3%/94.4%, i.e., much like the albumin dipstick results.The many reports on the association of albuminuria and risk of renal disease recommend that

screening should be done for albumin rather than protein. Based on the data from the school children, we estimate that a dipstick albumin of "30" mg/l is borderline increased risk, and that a protein dipstick of "150" mg/l is the same. If they call the dipstick "10" mg/l albumin, "30" mg/l albumin and the "150" mg/l protein results "low risk," then they estimate the prevalence of albuminuria in the school children to be about 2.1% and proteinuria to be about 4.3%. Children with these values should have a quantitative test for albumin and protein. They also tested a dipstick for creatinine and found increasing values with increasing age in both genders; the older boys had significantly higher creatinine values than the older girls and younger boys. For the albumin/creatinine ratio, we found 6028 children with a ratio of > or≥ 30 mg/g indicating low risk and 159 children with a ratio of > or = 30 mg/g indicating increased risk. The ratio may be more useful owing to the likely reduction of the number of false negative and false positives.

Screening urine for microhematuria as an indicator of serious disease is controversial because of the low positive predictive value of such screening and the costs and risks of the associated evaluation. To further evaluate test properties, **Hiatt RA, Ordonez JD. Division of Research, Kaiser Permanente Medical Care Program, Oakland, California 94611** retrospectively examined the outcomes of 20,571 men aged > or = 35 years and women aged > or = 55 years who voluntarily had a Personal Health Appraisal in 1980 as members of a large prepaid health plan. Hematuria was detected by dipstick in 876 cases (4.3%); 278 were excluded because of evidence of previous urological disease which could cause hematuria. Review of the medical records of 598 patients with asymptomatic microhematuria as shown by a positive dipstick result indicated that 99% had a follow-up evaluation within 3 months of positive

test results for hematuria and had various levels of urological evaluation thereafter. However, urological cancers (2 prostate, 1 bladder) developed in only 3 patients within the next 3 years. On the basis of San Francisco-Oakland Surveillance, Epidemiology, and End Results program data, rates of urological cancer were evaluated among patients whose test results were negative for hematuria, and these cancer rates were found to be almost the same as the rate among patients with asymptomatic microhematuria. Sensitivity of a single dipstick urinalysis result using microhematuria to indicate urological cancer within 3 years was 2.9%; specificity was 96.7%; and positive predictive value was 0.5%. Multivariate analysis which adjusted for age, gender, and race showed that the relative risk of 2.1 (95% confidence interval, 0.7-6.6) for urological cancer was not significantly elevated among patients with asymptomatic microhematuria compared with patients who had negative test results. These findings based on a single test are consistent with the current lack of recommendations for screening for microhematuria among asymptomatic adults.

The prevalence and incidence of renal diseases in developing countries are not known. This lack of knowledge is an obstacle to the adoption of preventive measures which may be of great value in a social and economic environment where treatment options for end-stage renal failure are simply not available to the vast majority of the population. Urinalysis, a simple and inexpensive test, remains a cornerstone in the evaluation of the kidney and may also be easily employed in mass screening for renal abnormalities in a developing country. **Plata R, Silva C, Yahuita J, Perez L, Schieppati A, Remuzzi G. Mario Negri per L'America Latina, Renal Diseases Project, Department of Nephrology and Dialisis Hospital Juan XXIII, La Paz, Bolivia.** Conducted an

educational campaign on renal diseases in three selected areas of Bolivia. Urine samples were collected and sent to one of 21 participating clinical centers. Fresh urine specimens were screened using a dipstick for chemical analysis and by microscopic urinalysis after centrifugation. In those patients in whom urinary abnormalities were found, further investigations were carried out in order to define the diagnosis; these patients were enrolled in a 3-year follow-up program. Apparently healthy subjects (n = 14,082) were referred to the First Clinical and Epidemiological Program of Renal Diseases from rural and metropolitan areas in Bolivia. Urinary abnormalities were detected in 4261 subjects at first screening. The most common form of urinary abnormality was hematuria, which was found in 2010 (47% of positively screened subjects). Other renal abnormalities were leukocyturia (41%) and proteinuria (11%). Confirmatory tests and further clinical studies were then carried out in 1019 people. On a second screening 35% of the subjects had no urinary abnormalities; in the remaining people the following diagnosis were made: asymptomatic urinary-tract infection (48.4%), isolated benign hematuria (43.9%), chronic renal failure (1.6%), renal tuberculosis (1.6%). Other diagnosis were: renal stones 1.3%, diabetic nephropathy 1% and polycystic kidney diseases 1.9%. CONCLUSIONS: This study helped define for the first time the frequency of asymptomatic renal diseases in Bolivia. It shows that it is possible to screen a large population of patients at relatively low cost, providing the framework for further action that may help in the prevention and timely diagnosis of renal diseases.

A possible method of improving the prognosis of bladder cancer may be the widespread introduction of screening. **Britton JP, Dowell AC, Whelan P, Harris CM. Department of Urology, St. Jame's University Hospital, Leeds, United Kingdom,** investigated the ability of urine

dipsticks to detect early bladder cancer in a group of men in the community. In 2,356 men more than 60 years old the urine was tested with a dipstick for the presence of blood. The subjects then tested their own urine on 10 subsequent occasions. Of the men 474 (20%) had dipstick hematuria and 319 agreed to undergo urological investigation. An asymptomatic bladder tumor was found in 17 men, associated in 10 with abnormal urine cytological findings. Urine dipsticks for the detection of red cells provided an inexpensive, simple and acceptable screening test for bladder cancer. However, introduction of generalized population screening by this method would produce large numbers requiring investigation. Combining urine cytology with dipstick hematuria results may provide a realistic alternative and further evaluation of the effectiveness of screening for bladder cancer in the community is required.

Microalbuminuria (MA) is associated with adverse health outcomes in diabetic and hypertensive adults. The prevalence and clinical significance of MA in nondiabetic populations is less clear. **Jones CA, Francis ME, Eberhardt MS, Chavers B, Coresh J, Engelgau M, Kusek JW, Byrd-Holt D, Narayan KM, Herman WH, Jones CP, Salive M, Agodoa LY. Division of Genetics and Epidemiology, Joslin Diabetes Center, Boston, MA, 02215, USA.** camille.jones@joslin.harvard.ed did a study to generate national estimates of the prevalence of MA in the US population. Untimed urinary albumin concentrations (UACs) and creatinine concentrations were evaluated in a nationally representative sample of 22,244 participants aged 6 years and older. Persons with hematuria and menstruating or pregnant women were excluded from analysis. The percent prevalence of clinical proteinuria (UAC > or = 300 mg/L) was similar for males and females. However, the prevalence of MA (urinary albumin-creatinine ratio [ACR], 30 to 299 mg/g) was significantly lower in

males (6.1%) compared with females (9.7%). MA prevalence was greater in children than young adults and increased continuously starting at 40 years of age. MA prevalence was greater in non-Hispanic blacks and Mexican Americans aged 40 to 79 years compared with similar-aged non-Hispanic whites. MA prevalence was 28.8% in persons with previously diagnosed diabetes, 16.0% in those with hypertension, and 5.1% in those without diabetes, hypertension, cardiovascular disease, or elevated serum creatinine levels. In adults aged 40+ years, after excluding persons with clinical proteinuria, albuminuria (defined as ACR > or = 30 mg/g) was independently associated with older age, non-Hispanic black and Mexican American ethnicity, diabetes, hypertension, and elevated serum creatinine concentration. MA is common, even among persons without diabetes or hypertension. Age, sex, race/ethnicity, and concomitant disease contribute to the variability of MA prevalence estimates. Copyright 2002 by the National Kidney Foundation, Inc.

To elucidate prognosis and prevalence of chronic renal diseases among proteinuric and/or hematuric subjects found in mass screening, a long-term follow-up study (6.35 years, range 1.03-14.6 years) was conducted on Japanese working men by **Yamagata K, Takahashi H, Tomida C, Yamagata Y, Koyama A. Institute of Clinical Medicine, University of Tsukuba, Japan. k-yamaga@md.tsukuba.ac.jp** A total of 772 subjects selected from 50,501 Japanese men aged 15-62 years were found to have asymptomatic hematuria (n = 404), concomitant hematuria and proteinuria (n = 155), and proteinuria (n = 213) during their annual urine examination and five consecutive urinalyses. Hematuria patients showed significant improvements in urinary abnormalities as compared with both hematuria/proteinuria and proteinuria patients. Both hematuria/proteinuria patients with normotension and

hematuria/proteinuria patients aged under 40 years showed significant improvements. During the follow-up period, 9.5% of the hematuria patients became hematuric/proteinuric. Hematuria/proteinuria patients had the highest risk of developing renal insufficiency. The presence of hypertension at detection of urinary abnormalities did not affect the renal function; however, if proteinuria appeared after the age of 40 years, these patients had a higher risk of developing renal insufficiency. The incidence of IgA nephropathy in the present subjects was as high as 143 cases per 1 million per year. Detailed follow-up and definitive diagnosis of asymptomatic urinary abnormalities may raise the prevalence of IgA nephropathy worldwide. Copyright 2002 S. Karger AG, Basel.

The incidence of asymptomatic bacteriuria is reported as 2-14% during pregnancy. Fetal and maternal complications like acute pyelonephritis, hypertension, anemia, preterm labor, low-birth-weight infants and intrauterine growth retardation can be expected. The purpose of this study was to determine the incidence of asymptomatic bacteriuria during pregnancy and its relation to pregnancy complications. The study involved 270 pregnant women up to 32 gestational weeks during a 9-month period. At the initial visit, they were screened with urine culture in order to detect asymptomatic bacteriuria. A control group was formed in a retrospective manner from the first day of the study with 186 pregnant women who delivered in our clinic and who were not screened for asymptomatic bacteriuria. The incidence of asymptomatic bacteriuria was 9.31%. Escherichia coli accounted for 79%, which was the most frequent of the isolates. We observed recurrence and had to apply treatment again to 21.7% of the women. The sensitivity, specificity, positive predictive and negative predictive values of leucocyturia as a screening test for asymptomatic bacteriuria were 91.3%, 83.6%, 45.6% and 98.5%,

respectively. We diagnosed preterm labor in six of 23 (26%) with asymptomatic bacteriuria and 16 in 163 (9.3%) women in the urine culture negative group. The ratio acute pyelonephritis in the group which was routinely screened and treated for asymtomatic bacteriuria was 0.5% while the prevalence was 2.1% in the nonscreened group. Considering the relatively high incidence of asymptomatic bacteriuria during pregnancy and the relevant complications, we propose to screen and treat asymptomatic bacteriuria routinely in all pregnant women.

MATERIAL & METHODS

MATERIAL & METHODS

To accomplish detection of asymptomatic urinary abnormalities in Bundelkhand region, it was necessary to screen the population in an effective and cheap way. Hospital was a biasing factor as most population of different regions draining into the hospital was symptomatic and suffering from variety of disease that may primarily or secondarily involved the renal system.

The best way was to conduct renal disease detection camps for the mass population in the study area with the help of health clubs and health promoting missionaries and projects.

The second problem was cost and effectiveness of the method used for screening. Eventually it was realized that uroscopy (cytobiochemical examination of urine) was a cheap and fruitful examination to screen for asymptomatic renal disease load in the population.

While screening the mass, a working format was decided for each individual attending the renal camps that included Name, Age, Sex, and Blood pressure, symptoms at presentation, urine examination, blood glucose examination and fundus examination. Individuals who had detectable urinary abnormalities were called the next day for repeat and further evaluation of the abnormalities and for reaching at a clinical diagnosis as the cause of this urinary abnormality. Of this mass of population screened a significant fraction was found to have detectable urinary abnormality and many of such patients were without clinical symptoms.

Taking the study conducted by Dr.V.N. Acharya in Bombay city as prototype, this study was conducted into 2 parts. First part comprised on initial assessment of urine (macroscopic and microscopic), blood pressure, and clinical symptoms of presenting patients and the second part was a follow up of cases that were detected to have urinary abnormality, so as to confirm the persistence of the urinary abnormality detected initially by repeat urine examination and work up and reach the cause of that persistent urinary abnormality.

For each part of this study simple working proforma were laid down and each patient that attended our renal camps was assessed according to this preplanned proforma.

WORKING PROFORMA 1 (Renal camps):

Included the following data for all group of people attending the renal camps :-

1. **Name :**

2. **Age :**

3. **Sex :**

4. **Blood pressure:** was recorded using mercury manometer with a 12 cm broad cuff. For children below 12 years an 8 cm duff was used. For correct recording the deflation rate was usually kept at 2mm per second.

5. **Clinical symptoms:** Any symptoms complained by the patient that appeared to be urological, were recorded. Patients with symptoms other than renal were considered to be asymptomatic.

6. Fresh Urine examination(Routine and Microcopy) :

COLLECTION:

There are 3 ways to obtain a urine specimen – spontaneous voiding, urethral catheterization, and suprapublic bladder puncture. Spontaneous voiding is the simplest and best method, if specimen is collected appropriately. A clean catch urine sample was obtained. In males, foreskin was retracted and glans penis cleansed. Similarly, in females, the labia were separated and area of labia and urethral meatus cleansed. Then midstream urine was collected .The present study used the spontaneous voiding method .

MACROSCOPIC EXAMINATION:

Urine sugar:-

In this study urine sugar was tested by benedict's method

BENEDICT'S TEST: - This test is based on the ability of sugar in urine to reduce cupric ions to cuprous ions. Though the test is not specific but it is better and more specific than the fehling's solution test.

Benedict's qualitative reagent – was prepared by dissolving 173 g of sodium citrate and 100 g of anhydrous sodium carbonate in about 600 ml warm water. This was filtered into a one liter volumetric flask.

Then 17.3 g of copper sulphate ($CuSO4.\ 5H_2O$) in about 100 ml water in a beaker and added slowly to the above mixture with constant shaking .This final mixture was made 1 litre by adding water.

Procedure: - 5 ml of Benedict's reagent was taken in tube and to it 0.4 ml (8 drops) of urine was added. The tube was heated directly on the flame till

the solution boiled. This was then cooled and examined for change of color.

Observation	Clinical record	Approx.glucose concentration (gram/100 ml urine)
Blue	0	Nil
Green(no precipitate)	±	Traces
Green(with precipitate)	+	0.5
Brown	+ +	1.0
Orange	+ + +	1.5
Red	+ + + +	2.0 and over

Note: - In addition to glucose the reduction of Benedicts solution may be caused by the following (false positive): (i) other sugars including lactate, fructose and pentoses. Lactose is the commonest, particularly during late pregnancy or lactation. (ii) Normal urine constituents, particularly uric acid, creatinine and ascorbic acid. Reduction is slight and only occurs with concentrated urines. (iii) The end products of drugs, commonly aspirin and salicylate (which are excreted as glucuronides and salicyluric acid).

Urine protein: – The presence of protein in urine is called proteinuria. Albumin is the main constituent, though higher molecular weight globulins also appear in the urine. Various simple methods for testing urine protein are available.

HEAT PRECIPITATION TEST- In this test the adjustment of urine pH to about 5 is necessary because the proteins only coagulate when they are heated at a pH near their isoelectric point (pH 5). False negative results may otherwise be obtained if the urine is alkaline or more acidic. Furthermore, by adjusting the pH before heating, the difficulties caused by the precipitation of phosphates can be avoided.

Reagents used :-

Acetic acid 33% solution.

Procedure – Take a test tube almost full of clear urine and test the pH with litimus paper or narrow range pH paper and adjust by drop wise addition of 33 percent acetic acid until it is slightly acidic (about pH5) . Heat the top few centimeters of the urine column to boiling and note any turbidity by comparison with the unheated part of the liquid. Appearance of turbidity or precipitate confirms the presence of protein.

MICROSCOPIC EXAMINATION:

Preparation of urine sediment is the first step in microscopic analysis. The importance of standardization of technique and quality accuracy cannot be over stressed to ensure accurate and reproducible analysis. Important steps include centrifugation, resuspension of sediment, slide preparation, and microscopic examination. In brief 10 ml of urine was centrifuged at approximately 2,000rpm (1,000-2,500rpm) in a centrifuge machine for 5 minutes. The supernatant 9ml was discarded and sediment was resuspended in 1ml. A drop of this was pipette onto a slide and a cover slip placed. The slide was then examined without staining. The slid was examined both under light and high power field in the microscop the following:

a). **Pus cells** : A value of more than 6 pus cells / HPF was c significant in this study.

HEAT PRECIPITATION TEST- In this test the adjustment of urine pH to about 5 is necessary because the proteins only coagulate when they are heated at a pH near their isoelectric point (pH 5). False negative results may otherwise be obtained if the urine is alkaline or more acidic. Furthermore, by adjusting the pH before heating, the difficulties caused by the precipitation of phosphates can be avoided.

Reagents used :-

Acetic acid 33% solution.

Procedure – Take a test tube almost full of clear urine and test the pH with litimus paper or narrow range pH paper and adjust by drop wise addition of 33 percent acetic acid until it is slightly acidic (about pH5) . Heat the top few centimeters of the urine column to boiling and note any turbidity by comparison with the unheated part of the liquid. Appearance of turbidity or precipitate confirms the presence of protein.

MICROSCOPIC EXAMINATION:

Preparation of urine sediment is the first step in microscopic analysis. The importance of standardization of technique and quality accuracy cannot be over stressed to ensure accurate and reproducible analysis. Important steps include centrifugation, resuspension of sediment, slide preparation, and microscopic examination. In brief 10 ml of urine was centrifuged at approximately 2,000rpm (1,000-2,500rpm) in a centrifuge machine for 5 minutes. The supernatant 9ml was discarded and sediment was resuspended in 1ml. A drop of this was pipette onto a slide and a cover slip placed. The slide was then examined without staining. The slid was examined both under light and high power field in the microscop the following:

a). **Pus cells** : A value of more than 6 pus cells / HPF was c significant in this study.

b). **RBCs**: A value of more than 5 RBCs / HPF was considered significant in this study.

c). **Crystals**: presence of any number or variety of crystals was considered as urinary abnormality in this study

6. Blood glucose:

Depending upon clinical symptoms and urine examination results, a blood glucose examination was done where needed. Conditions like, presence of sugar or protein, or pus cells in urine, presence of hypertensive and symptom like obesity, polyuria, nocturnal, polydipsia all demanded a blood glucose examination and so it was conducted in all such cases.

7. Fundoscopy: Fundus was examined in all cases having diabetes mellitus or hypertension to check diabetic retinopathy or vessel changes suggestive of hypertensive.

WORKING PROFORMA 2 (follow up):

This included at our hospital the following examination and information.

1. Name:

2. Age:

3. Sex:

4. Repeat Blood pressure:

5. Clinical Symptoms: Presence of clinical symptoms suggestive of renal disease were asked for and signs for renal disease looked for.

6. Repeat urine(routine/microscopy) : Same method was used as used in the working proforma 1.

7. Serum creatinine :

Principle : Creatinine is treated with picric acid in alkaline medium , a red colour develops which is measured colorimetrically. The reaction is not specific but at least over 85 percent colour is due to creatinine

Reagents used :-

-Sodium tungstate 10 %

-Sulphuric acid (2/3 N)

-Sodium hydroxide, 10 percent

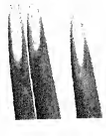
-Saturated picric acid solution

-Stock creatinine standard – Dissolve 100mg of pure dry creatinine in 100mg of 0.1 N-HCl

-Working creatinine standard-Dilute 1.0 ml stock solution to 10 ml with water.

-Alkaline picrate solution- Prepare just before use a mixture of 10 ml saturated picric acid and 2 ml sodium hydroxide .

Procedure:-



Test – in a centrifuge tube 1.0 ml serum was taken. 4 ml water and 0.5 ml sodium tungstate and 0.5 ml sulphuric acid was added to the serum. Solution was mixed by inversion and centrifuged after some time. 3 ml supernatant was taken in another tube.

Standard – 3 ml working standard

Blank – 3 ml water.

1.5 ml alkaline picrate solution was added to each tube. Mixed well and allowed to stand for 10 minutes. The absorbance using green filter (520 nm) against the blank was measured. Calculation :

$$\text{Serum creatinine} = \frac{T}{S} \times 6 \text{ (mg/100ml)}$$

The normal range of serum creatinine is 0.1 to 1.2 mg/100ml.

Increased values are usually found in advanced cases of renal disease. With severe renal failure it may rise to over 10 mg/100ml.

8. Repeat Blood Glucose (Fasting): Repeat blood glucose was done. This value was fasting as all cases called for follow up were told to stay 10 hour empty stomach before coming for follow up.

9. Urine Culture: Was done in all patients found to have pus cells in their urine. A clean catch sample was collected in a test tube and a loopful was inoculated on 2 types of media for 24 hours. The 2 media used were MacConkey's agar and Blood agar . Growth was observed for and interpreted as follows: More than 10^5 organisms per ml indicated definite infection from that species, between 10^2 and 10^5 organisms indicated possible infection and less than 10^2 organisms from a single strain excluded infection .

10. Renal biopsy: Biopsy was not done in all patients found to have urinary abnormalities. It was only done in cases where the diagnosis was not clear. It was done in the morning after an overnight fasting. An i.v. access was established and maintained for the next 24 hours. Prior to the biopsy the biopsy sight was localized by USG. The Patient was made to lie in a proned position and skin cleaned, draped and infiltrated with local anesthetic agent. A seven inches needle (mostly lumbar puncture needle) was used to explore the kidney (movement of the needle was looked for during respiration).the biopsy needle used by in this study was a true cut needle (11.4cm). After adjusting the length of the true cut needle the biopsy was done. Adequacy of the sample was checked. If inadequate sample, the procedure was repeated. After taking adequate sample, it was transferred into a formalin (10 %) solution in a test tube and send for histopathology and immunoflourescent study. Results were usually obtained within 2-3 days. Care was taken in regards to the patient for the next 24 hours and features like post biopsy hematuria and hypertension and hypotension were specially monitored for in these 24 hours

11. Final diagnosis: Using above mentioned methods and procedures cases detected to have asymptomatic urinary abnormalities were diagnosed of their underlying disease.

OBSERVATION

OBSERVATION

There were 279 patients seen in various renal camps, held in the region of Bundelkhand and their distribution was as follows :-

Table No I (CONSTITUENT POPULATION IN STUDY)

Age Range(years)	Males	Females	Total
0-12 Yrs	09	16	25
12-20 Yrs	13	14	27
21-40Yrs	81	49	130
41-60Yrs	53	24	77
Above 60 yr	16	04	20
Total	172	107	279

As shown in table No. 1 a total of 279 people were included in this study. Out of these 279 people the male population was 172 patients making 61.65% of study population and there were 107 females making 38.35% of the study population.

Population upto 12 year of age (paediatric group) included 25 people that is 8.96% of the total, in which there were 9 males (3.22%) and 16 females (5.73%).

The male is to female ratio as a whole in the study population was 1.6:1.

Maximum number of people attending the Renal camp and turning up for re-evaluation were of the age group 21-40 year and minimum number belonged to the age group above 60 years.

Table II (A)

URINARY ABNORMALITY DETECTED IN THE STUDY POPULATION

Age group (years)	No urinary abnormality	Urinary abnormality detected	Total	% with urinary abnormality
0-12	19	06	25	24%
13-20	23	04	27	14.81%
21-40	104	26	130	20%
41-60	53	24	77	31.16%
61 and above	12	08	20	40.00%
Total	211	68	279	24.37%

As a whole it was found, that there were 68 patients out of 279 screened who has detectable urinary abnormalities both during initial screening and follow up study. This made up about 24.37% of the total. Considering different age groups it was 24% in age group 0-12 ; 14.8% in age group 13-20 ; 20% in Age group 21-40% ; 31.16% in age group 41-60 and 40% in age group above 61 years and above. The maximum percentage of urinary abnormality detected was in the age group 61 years and above (40%) and the minimum percentage was for the age group 13-20 years (14.81%).

TABLE IIB

PATIENTS HAVING ASYMPTOMATIC URINARY ABNORMALITIES

Age group (years)	Symptomatic Or no urinary abnormality	Asymptomatic Urinary abnormality	Total	% of Asymptomatic urinary abnormalities
0-12	21	04	25	16%
13-20	24	03	27	11.11%
21-40	108	22	130	16.92%
41-60	62	15	77	19.48%
≥ 61	14	06	20	30.00%
Total	229	50	279	17.9%

As seen in table II B, out of 279 patients screened, 50 patients were detected to have asymptomatic urinary abnormalities, as compared to 68 patients detected to have urinary abnormalities (symptomatic or asymptomatic). This was 17.9% of the total population screened.

For different age groups, the distribution of asymptomatic urinary abnormalities varied, being maximum in the age group above 61 years (30.00%) and minimum in the age group 12 – 20 years(11.11%) , the percentage of other age groups lying between these two as shown in the above table .

Table III.

SEX DISTRIBUTION OF ASYMPTOMATIC URINARY ABNORMALITIES DETECTED

Age Group	Male (no. of cases)			Female (no. of cases)		
	No Urinary Abnormalities	Urinary Abnormalities	%	No Urinary Abnormalities	Urinary Abnormalities	%
0-12	08	01	12.50%	13	03	18.75%
13-20	12	01	7.60%	12	02	14.28%
21-40	67	14	17.28%	41	08	16.32%
41-60	45	08	15.09%	17	07	29.16%
61-above	11	05	31.25%	03	01	25%
Total	143	29	20.27%	86	21	24.41%

The male to female ratio for total and asymptomatic urinary abnormalities was as shown in Table III

(1) For the age group 0-12 years :- Asymptomatic Urinary abnormalities were detected in 12.5% of males and 18.75% of females.

(2) For Age group 13-20 year:-Asymptomatic Urinary abnormalities were detected in 7.6% of males and 14.28% of females.

(3) In Age group 21-40 years:- Asymptomatic Urinary abnormalities were detected in 17.28% of males and 6.32% of females.

(4) In Age group 41-60 years:-Asymptomatic Urinary abnormalities were detected in 15.09% of males and 29.16% of females.

(5) In Age above 60 years:-`Asymptomatic urinary abnormality was seen in 31.25% of males and 25% of females.

DIFFERENT URINARY ABNORMALITIES DETECTED

(A) PROTIENURIA

Prevalence of Asymptomatic Proteinuria

TABLE NO. VI

PATIENTS HAVING ASYMPTOMATIC PROTEINURIA AND ISOLATED PROTEINURIA

Age group (yr)	Total Asymptomatic Urinary Abnormalities Detected	asymptomatic Proteinuria (%)	Isolated Proteinuria (%)
0-12	04	02(50%)	01(25%)
13-20	03	00(0%)	00(0%)
21-40	22	06(27%)	06(27.27%)
41-60	15	08(27.27%	06(40%
61& above	06	03(50%	01(16.66%
Total	50	19(38%)	14(28%)

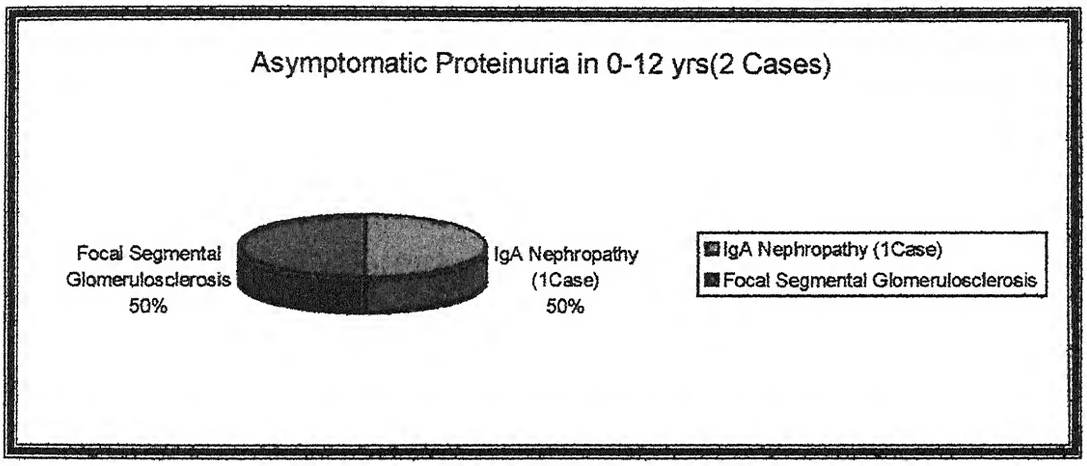
Of the various urinary abnormalities detected, proteinuria was a commonly detected abnormality. It occurred either as isolated proteinuria or along with other abnormalities like pyuria, RBCs, or oxalate crystal etc.

In the age group of 0-12 yrs asymptomatic proteinuria constituted 50%, of which isolated proteinuria constituted 25% of all asymptomatic urinary abnormalities for this age group. Proteinuria was not detected in the age group 13-20 years. Again in age group 21-40yrs proteinuria constituted 27.27% of asymptomatic urinary abnormalities for that age group out of which all were isolated proteinuria. For age group 41-60yrs proteinuria constituted 53.33% of which isolated proteinuria was 40% of all asymptomatic urinary abnormalities for the age group. In 61 years and above, proteinuria was 30% of which isolated proteinuria was 16.66% of all sympatric urinary abnormalities for that age group. Out of 50 asymptomatic

urinary abnormalities 19 were proteinuria of which 14 were isolated proteinuria. Thus Proteinuria constituted 38% of all asymptomatic urinary abnormalities and isolated proteinuria constituted 28% of all asymptomatic urinary abnormalities.Thus out of the study population of 279 people screened asymptomatic proteinuria was found in 6.81% of people (19 cases) and isolated proteinuria was found in 5.02% people (14 cases).

Causes of Asymptomatic Proteinuria

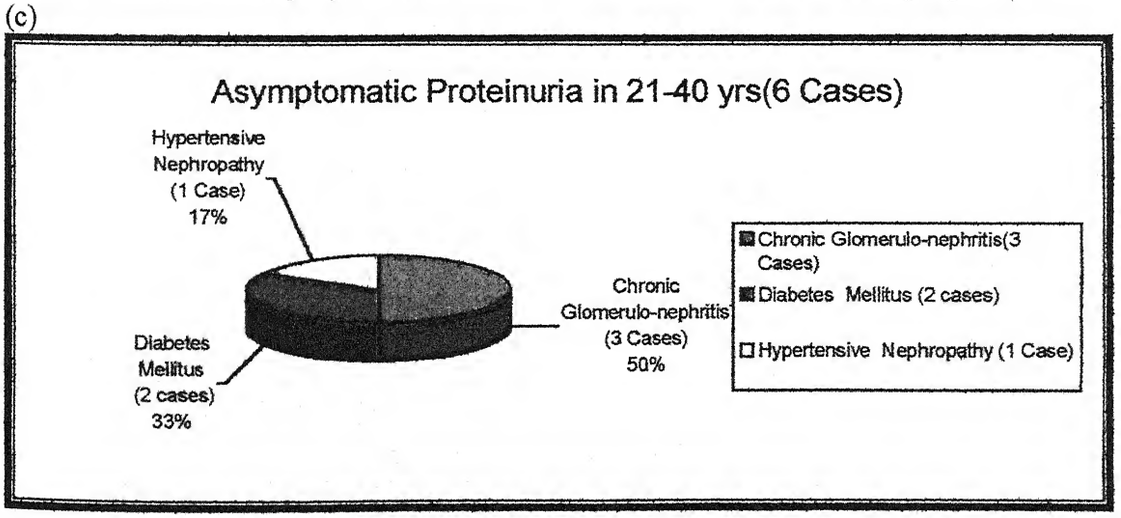
(a)



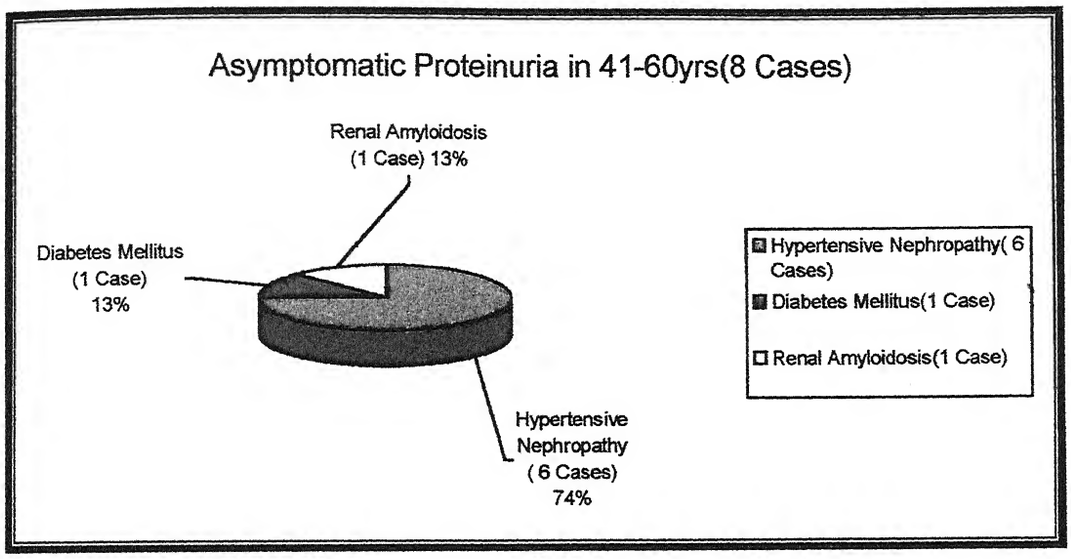
(b)

Asymptomatic proteinuria in 13-20 yrs (0 cases)
No cases of Asymptomatic Proteinuria were seen in this age group

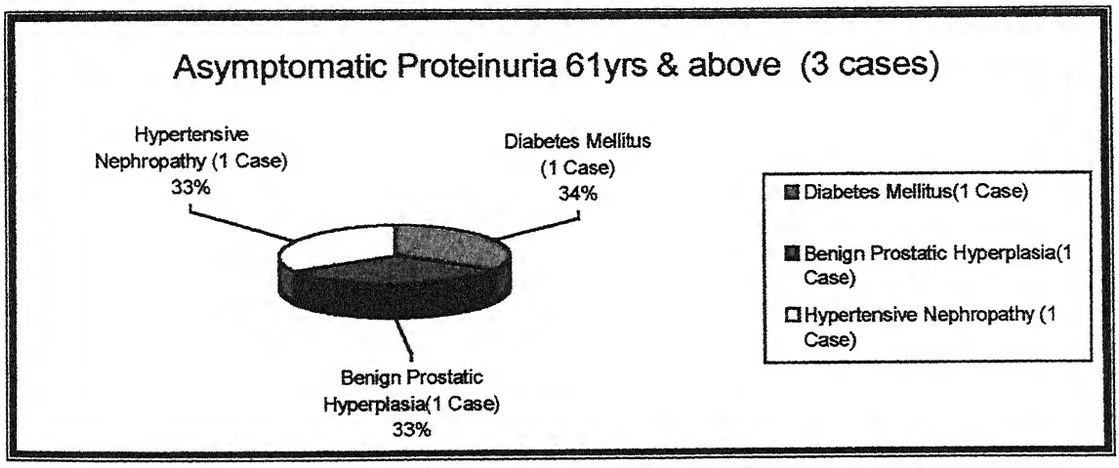
(c)

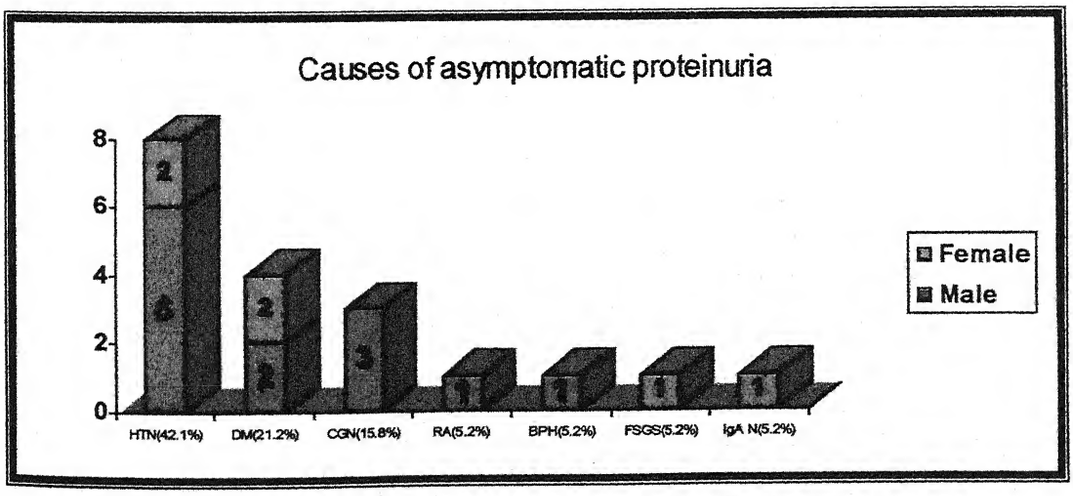


(d)


Asymptomatic Proteinuria in 41-60yrs(8 Cases)
Renal Amyloidosis
(1 Case) 13%
Diabetes Mellitus
(1 Case)
13%
Hypertensive
Nephropathy
(6 Cases)
74%
Hypertensive Nephropathy(6 Cases)
Diabetes Mellitus(1 Case)
Renal Amyloidosis(1 Case)

(e)


Asymptomatic Proteinuria 61yrs & above (3 cases)
Hypertensive
Nephropathy (1 Case)
33%
Diabetes Mellitus
(1 Case)
34%
Benign Prostatic
Hyperplasia(1 Case)
33%
Diabetes Mellitus(1 Case)
Benign Prostatic Hyperplasia(1 Case)
Hypertensive Nephropathy (1 Case)


Causes of asymptomatic proteinuria
8
6
4
2
0
HTN(42.1%)
DM(21.2%)
CGN(15.8%)
RA(5.2%)
BPH(5.2%)
FSGS(5.2%)
IgA N(5.2%)
Female
Male

As shown in the above table it was seen that the major causes of asymptomatic proteinuria was Hypertensive nephropathy (42.1%) , Diabetes mellitus (21.2%) , and Chronic glomerulonephritis (15.8%) , other causes being Renal amyloidosis , Benign prostatic hyperplasia , Focal segmental glomerulosclerosis , and IgA Nephropathy .

(B) ASYMPTOMATIC PYURIA

Prevalence of Asymptomatic Pyuria

Table No. V

PATIENTS DETECTED HAVING ASYMPTOMATIC PYURIA

Age Group	Total Asympt. Urinary Abnormalities	Asymptomatic Pyuria with other abnormalities	Asymptomatic Isolated Pyuria
0-12	04	01(25%)	01(25%)
13-20	03	03(100%)	03(100%)
21-40	22	11(50%)	07(31.8%)
41-60	15	07(46.66%)	05(33.33%)
61& above	06	04(66.66%)	01(16.66%)
Total	50	26(52.00%)	17(34%)

Another urinary abnormality commonly encountered in this study was pyuria. Pyuria was detected very commonly and more so in young females. The male to female ratio in all asymptotic cases of pyruia detected was 14:12. For age group 0-12 years 25% of all asymptomatic urinary abnormalities was pyuria of which all was isolated pyuria. For age group 13-20, the prevalence was 100% of all asymptomatic abnormalities (all asymptomatic urinary abnormalities were isolated pyuria). For age groups 21-40 years prevalence of pyuria was 50% of which isolated pyuria was 31.8% of all asymptomatic urinary abnormalities for this group. For age group 41 to 60 asymptomatic pyuria with other abnormalities constituted 46.66% & isolated pyuria constituted 33.33% of all

asymptomatic urinary abnormalities.For age groups 61 years and above asymptomatic pyuria with other abnormalities constituted 66.66% & isolated asymptomatic pyuria constituted 16.66% of all asymptomatic urinary abnormalities.As a whole asymptomatic pyuria constituted 26 cases out of 50 asymptomatic urinary abnormalities in this study constitute 52% of all asymptomatic urinary abnormalities and isolated asymptomatic pyuria constituted 17 cases of 50 asymptomatic urinary abnormalities constituted 34% of all asymptomatic urinary abnormalities.

Causes of asymptomatic pyuria

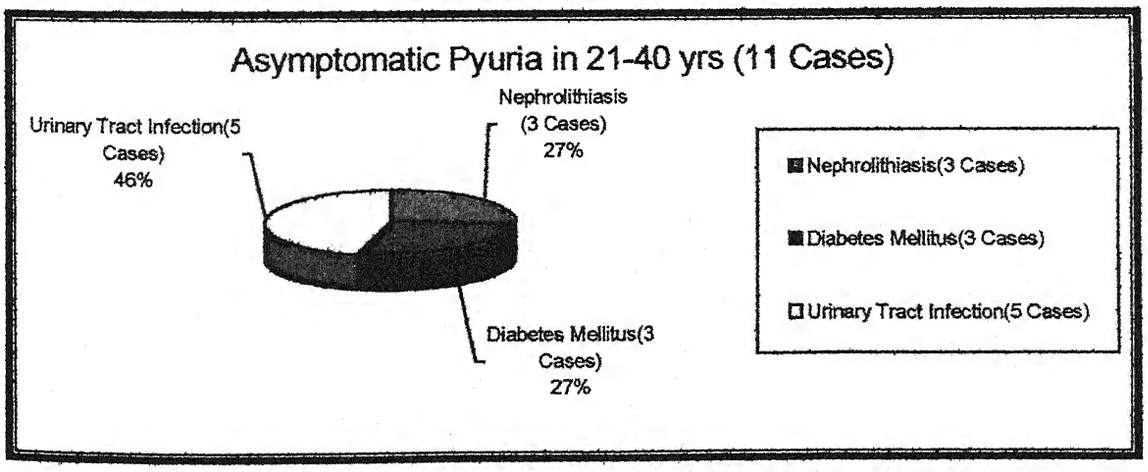
(a) Asymptomatic pyuria in 0-12 yrs (1 case)

In 0-12 yrs only one Male case of Asymptomatic Pyuria was detected and that was due to vesico ureteral reflux disease.

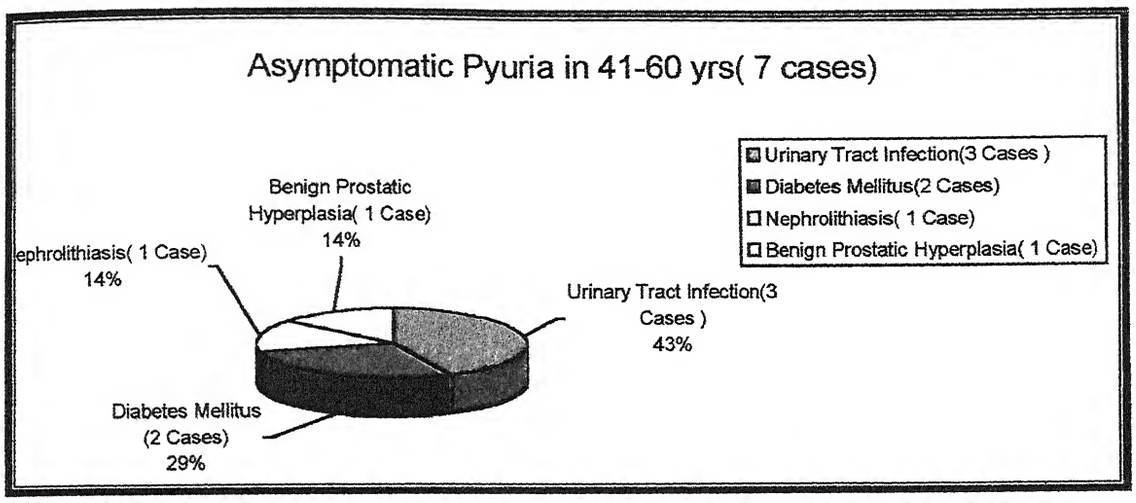
(b) Asymptomatic pyuria in 13-20 yrs (3 case)

There were 3 cases in this age group and all were due to urinary tract infection (1 was male, 2 were females)

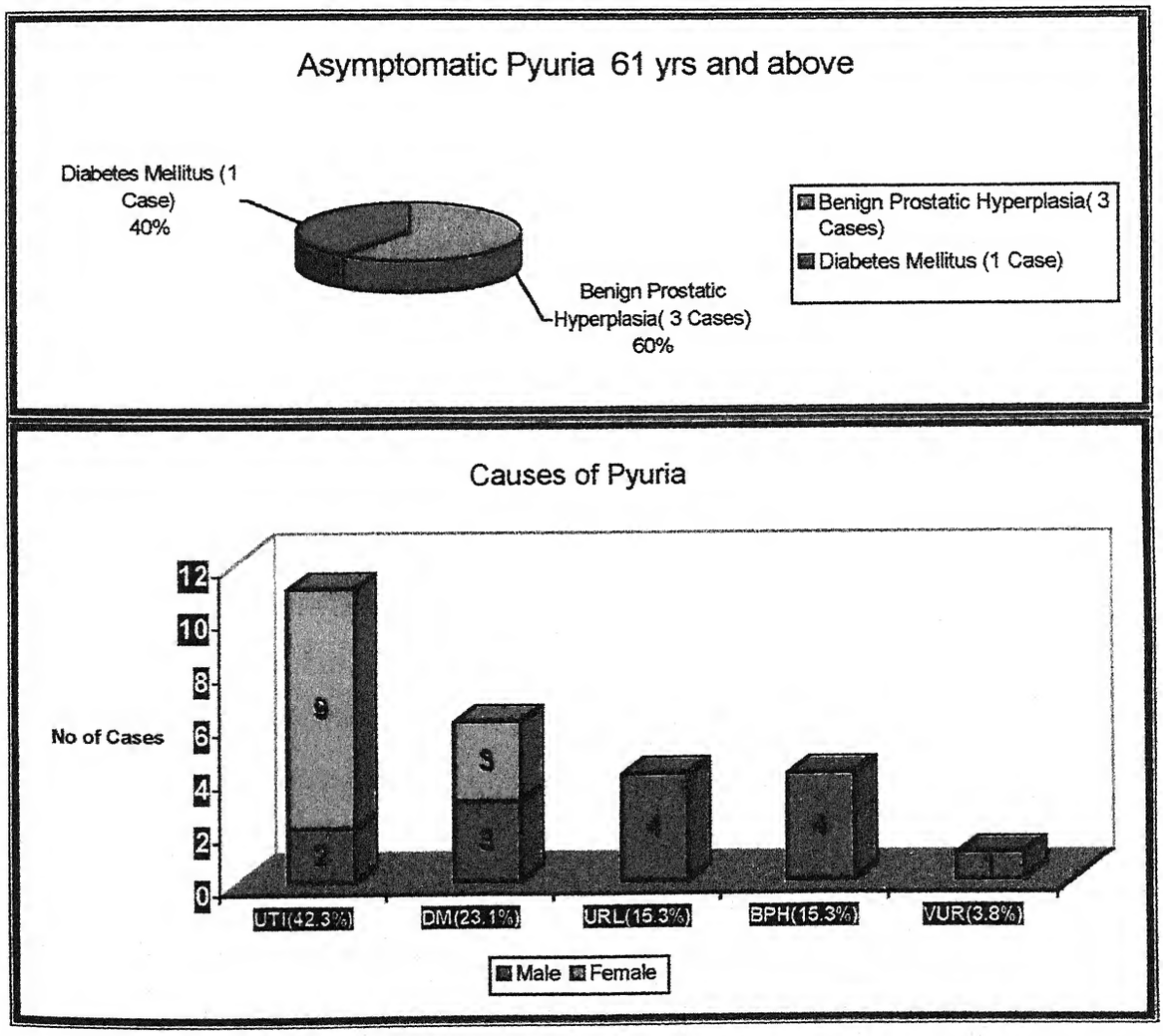
(c)



(d)


Asymptomatic Pyuria in 41-60 yrs(7 cases)
Urinary Tract Infection(3 Cases)
Diabetes Mellitus(2 Cases)
Nephrolithiasis(1 Case)
Benign Prostatic Hyperplasia(1 Case)
Benign Prostatic Hyperplasia(1 Case) 14%
ephrolithiasis(1 Case) 14%
Urinary Tract Infection(3 Cases) 43%
Diabetes Mellitus (2 Cases) 29%

(e)


Asymptomatic Pyuria 61 yrs and above
Diabetes Mellitus (1 Case) 40%
Benign Prostatic Hyperplasia(3 Cases) 60%
Benign Prostatic Hyperplasia(3 Cases)
Diabetes Mellitus (1 Case)
Causes of Pyuria
No of Cases
0
2
4
6
8
10
12
9
2
5
3
4
4
1
UTI(42.3%)
DM(23.1%)
URL(15.3%)
BPH(15.3%)
VUR(3.8%)
Male
Female

As shown in the above bar diagram , the causes of asymptomatic pyuria were urinary tract infection (42.3% , mostly females) , diabetes mellitus (23.1% with equal male female ratio) , urolithiasis (15.3% only in males) , benign prostatic hyperplasia (15.3% only in males) and vescicu ureteral reflux (3.8%only in males).

(C) ASYMPTOMATIC GLYCOSURIA

Prevalence of Asymptomatic Glycosuria

Table No. VI

PATIENTS DETECTED HAVING ASYMPTOMATIC GLYCOSURIA

Age Group	Total Urinary Abnormalities	Glycosuria (asymptomatic)	Isolated Glycosuria (asymptomatic)
0-12	04	01(25%)	01(25%)
13-20	03	00(0%)	00(0%)
21-40	22	03(13.6%)	010(4.5%)
41-60	15	02(13.33%)	02(13.13%)
61& above	06	02(33.33%)	00(0%)
Total	50	08(16%)	04(8%)

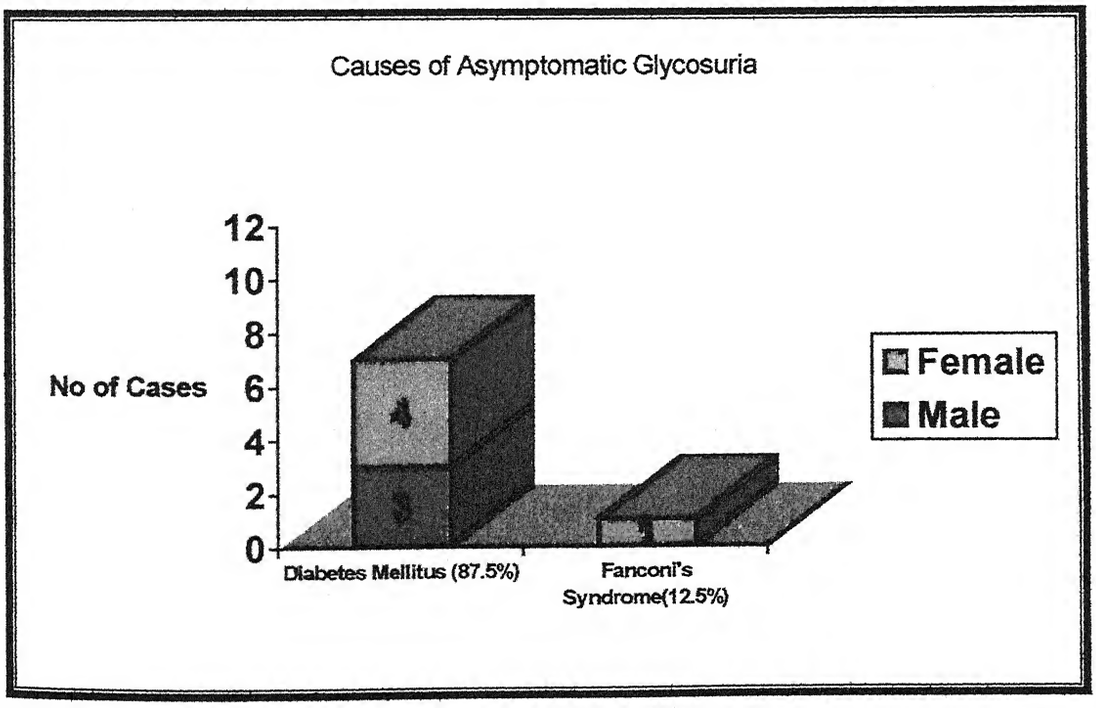
Asymptomatic Glycosuria occurred as isolated Glycosuria & as Glycosuria along with other urinary abnormalities, out of 50 cases of asymptomatic urinary abnormalities total case of asymptomatic Glycosuria were 8 making it 16% of all asymptomatic urinary abnormalities of which 4 were asymptomatic isolated glycosuria making it 5% of all asymptomatic urinary abnormalities.For different age group the prevalence of glycosuria with other urinary abnormalities and isolated glycosuria was as follows: For 0-12 Asymptomatic Glycosuria constituted 25% of all asymptomatic urinary abnormalities for that age group of which all occurred as isolated glycosuria. In age group 13-20 years no glycosuria was detected. For group 21-40 years Asymptomatic Glycosuria constituted 13.6% of all asymptomatic urinary abnormalities for that age group of which isolated glycosuria was 4.5 %. For group 41-60 years Glycosuria occurred only as

isolated glycosuria in this age group and prevalence was 13.13% of all of all asymptomatic urinary abnormalities for that age group. For above 61 - Glycosuria occurred only as glycosuria with other abnormalities & prevalence was 33.35%.

Causes of Asymptomatic glycosuria

Age Group	Causes	Sex distribution
0-12	1 fanconi's syndrome	Female
13-20	0	
21-40	3DM	2 Male, 1 Female
41-60	2 DM	Both Females
61& above	2 DM	1 Male , 1 Female

All cases asymptomatic glycosuria / asymptomatic isolated glycosuria were attributed to Diabetic Mellitus except for 1 case which was due to fanconi's syndrome in the age group 0 – 12 years in this study the ratio of male: female was 3:4 respectively.



As shown in the above bar diagram , the major cause of asymptomatic glycosuria was diabetes mellitus (87.5% with slight female

predominance) with fanconi's syndrome making a small percentage (12.5% all females).

(D) ASYMPTOMATIC CRYSTALLURIA

Prevalence of Asymptomatic Crystalluria

Table No. VII

PATIENTS DETECTED HAVING ASYMPTOMATIC CRYSTALLURIA

Age group	Total urinary abnormalities	Asymptomatic Crystalluria	Asymptomatic Isolated Crystalluria
0-12	04	00(0%)	00 (0%)
13-20	03	00(0%)	00(0%)
21-40	22	05(22.72%)	03(13.63%)
41-60	15	01(6.66%)	00(0%)
61& above	06	00(0%)	00(0%)
Total	50	06(12%)	03(6%)

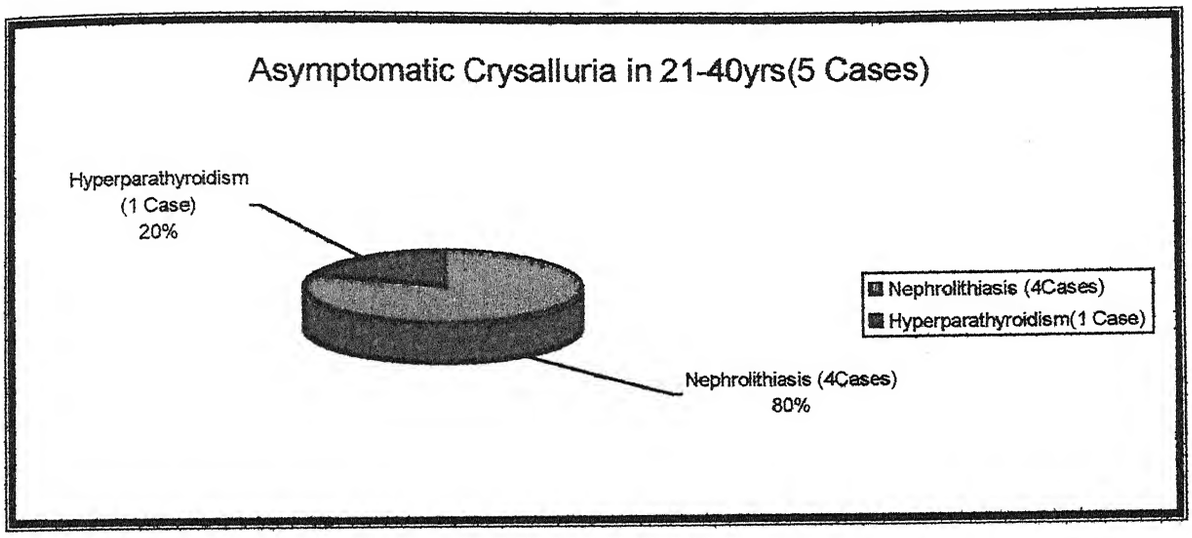
There were a total 6 cases of crystalluria and 3 cases of isolated crystalluria out of 50 asymptomatic urinary abnormalities , making them 12% & 6% respectively . No cases were seen in age group 0-12 & 13-20 year . Maximum 5 cases of asymptomatic crystalluria were seen in age group 21-40 years making it 22.72% of asymptomatic urinary abnormalities of that age group , out of which 13.63% occurred as isolated asymptomatic crystalluria . For age group 41-60 prevalence of asymptomatic crystalluria was 6.66% of all urinary abnormalities for that age group.

Causes of Asymptomatic crystalluria

(a) Asymptomatic Crystalluria 0-12yrs (0 cases)

(b) Asymptomatic Crystalluria 13-20 yrs (0 cases)

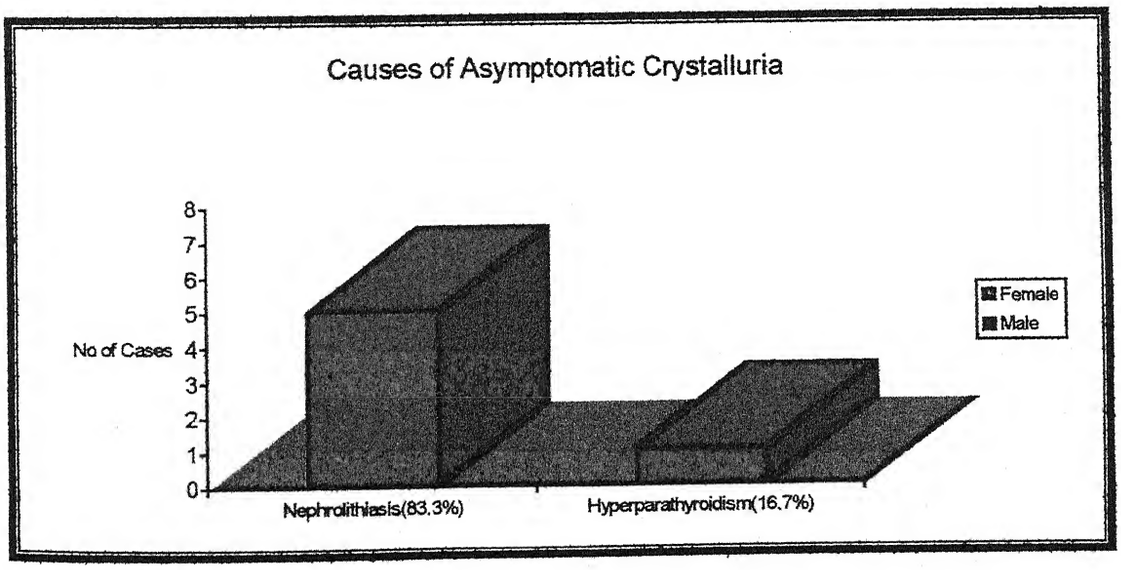
(c)



(d) Asymptomatic Crystalluria 41-60 yrs (1 cases)

One case of Asymptomatic Crystalluria (Male) was detected and this was caused by nephrolithiasis.

(e) Asymptomatic Crystalluria 61 yrs and above(0 cases)



As shown in the above bar diagram, the major cause of asymptomatic Crystalluria was Nephrolithiasis (83.3% all being males) while another cause hyperparathyroidism was also found (16.7% all cases being males).

(E) HEMATURIA

Prevalence of Asymptomatic hematuria

TABLE No. VIII

PATIENTS DETECTED HAVING ASYMPTOMATIC HEMATURIA

Age group	Total urinary abnormalities	Hematuria	Isolated Hematuria
0-12	04	01 (25%)	00 (0%)
13-20	03	00 (0%)	00 (0%)
21-40	22	02 (9.09%)	01 (4.54%)
41-60	15	01 (6.66%)	00 (0%)
61& above	06	01 (16.66%)	00 (0%)
Total	50	05 (10%)	01 (2%)

Causes of Asymptomatic hematuria

(a) Asymptomatic Hematuria in 0 – 12 years(1 Case)

Only one case (female) was detected in this group and the cause was IgA Nephropathy.

(b) Asymptomatic Hematuria in 13 – 20 years(0 Case)

(c) Asymptomatic Hematuria in 21 – 40 years(2 Case)

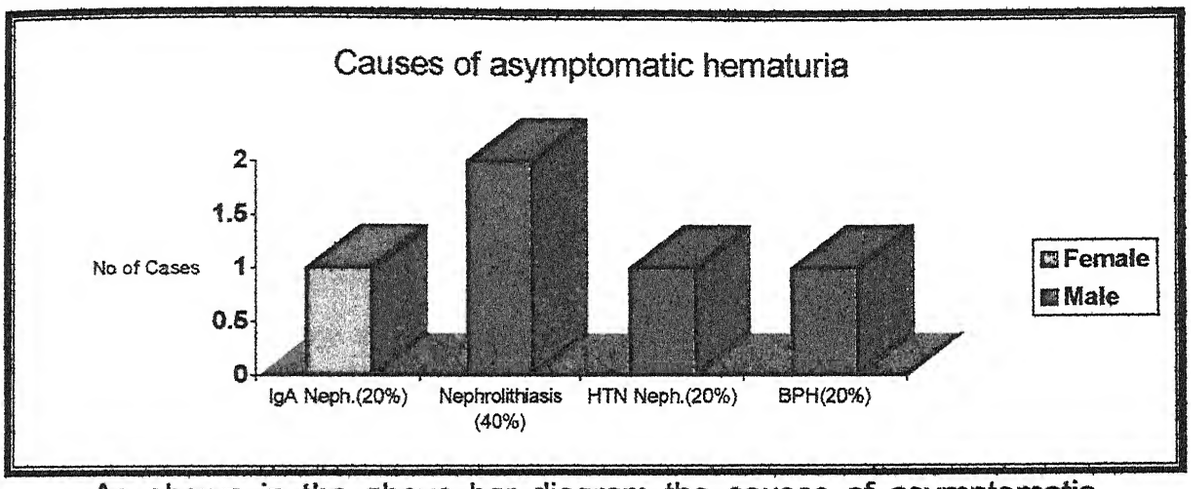
Both cases of asymptomatic hematuria in this group were males and both were caused by nephrolithiasis

(d) Asymptomatic Hematuria in 41 - 60years(1 Case)

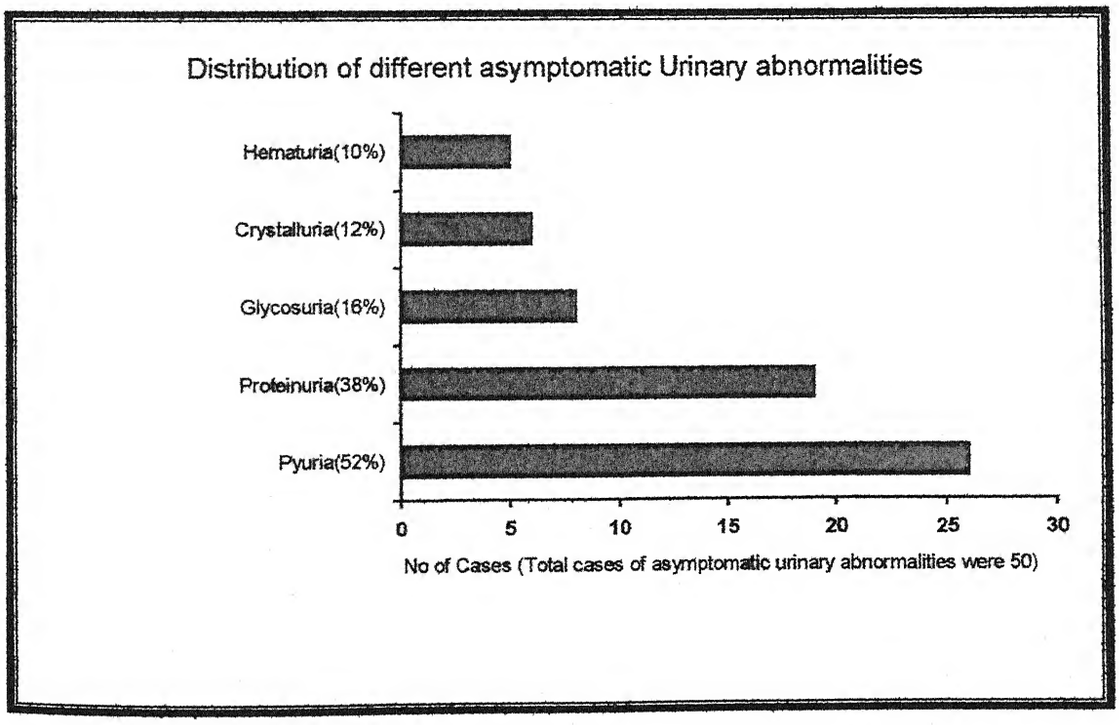
This male case of hematuria was due to Hypertensive Nephropathy.

(e) Asymptomatic Hematuria in 61 years and above(1 Case)

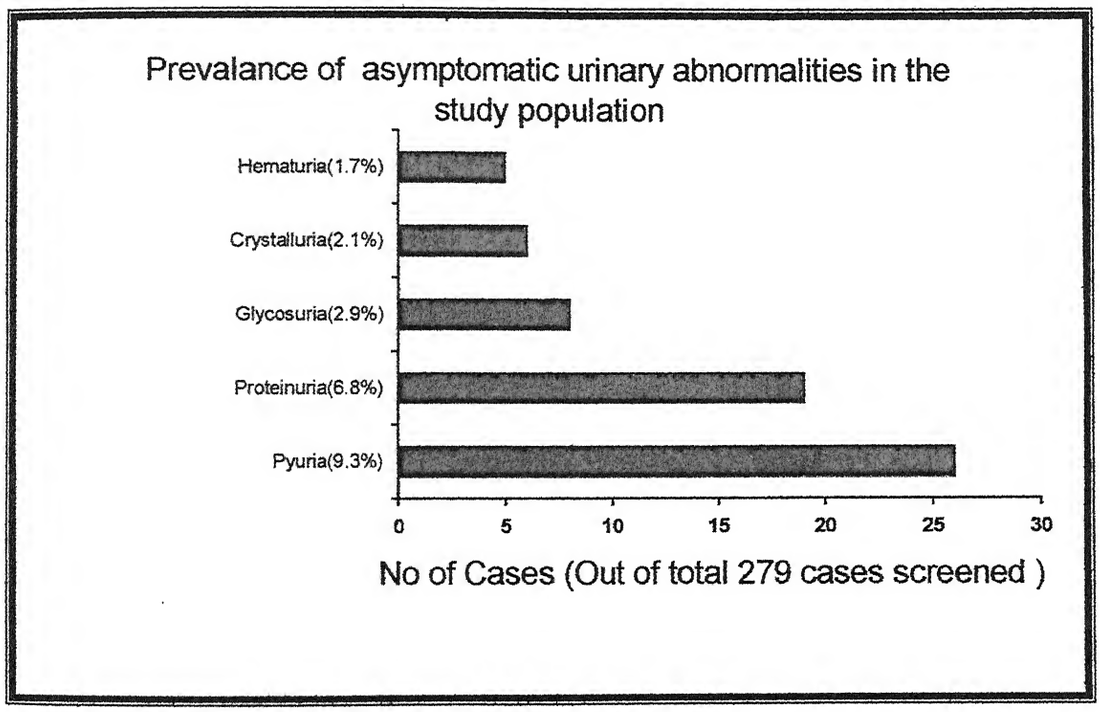
There was only one case of hematuria in this group and this was due to benign prostatic hypertrophy.



As shown in the above bar diagram the causes of asymptomatic hematuria were Nephrolithiasis (40%) , IgA Nephropathy(20%), hypertensive Nephropathy (20%) and benign prostatic hyperplasia (20 %).



Amongst all different types of asymptomatic urinary abnormalities detected, asymptomatic Pyuria had the most common prevalence (52%), followed by asymptomatic Proteinuria(38%), Asymptomatic Glycosuria had prevalence of 16% , asymptomatic Crystalluria was 12% and asymptomatic Hematuria was 10%.



As shown in this chart above , out of 279 cases screened pyuria was seen in 9.3% cases, proteinuria was seen in 0.3%cases , glycosuria was seen 2.9%cases , crystalluria was seen in 2.1% cases and hematuria was seen in 1.7%cases .

PREVALENCE OF ASYMPTOMATIC RENAL DISEASE

TABLE NO. I

AGE AND SEX DISTRIBUTION OF ASYMPTOMATIC DISEASES

Age group	Asymptomatic diseases detected (No of Cases)	Sex distribution
0-12	1 Fanconi's syndrome 1 Vesico ureteral reflux 1 FSGS 1 IgA Nephropathy	1 female 1 male 1 female 1 female
13-20	3 UTI	2 Female , 1 male
21-40	6 NPL (all males), 6 DM 5 UTI 3CGN	All males 3 Male, 3 Females 5 Females 3 Males
41-60	1 NPL 5 DM 6 HTN Nephropathy 3 UTI 1 BPH 1 Renal Amyloidosis 1 Hyperparathyroidism	1 Male 2 Males ,3 Females 4 Males, 2 Females 1 Male , 2 Female 1 Male 1 Male 1 Male
61& above	2 DM 3 BPH 1 HTN Nephropathy	1 Male , 1 Female 3 Males 1 male

As shown in table no. 1, there were various types of asymptomatic urinary abnormalities detected in different age groups, whose number and sex distribution varied with each age group . The above table shows age and sex distribution of all 50 cases of asymptomatic urinary abnormalities in this study.

TABLE NO. II

PREVALENCE OF ASYMPTOMATIC DISEASES IN THIS STUDY

Disease	No. of asymptomatic cases detected	Ratio M:F	% of all asymptomatic Urinary abnormalities	% of total population screened
Diabetes Mellitus	13	6:7	26%	4.65%
Urinary tract infection	11	2:9	22%	3.94%
Nephrolithiasis	07	7:0	14%	2.51%
Hypertensive Nephropathy	07	5:2	14%	2.51%
Chronic Glomerulo Nephritis	03	3:0	6%	1.07%
Benign prostatic hyperplasia	04	4:0	8%	1.43%
Renal Amyloidosis	01	1:0	2%	0.35%
Hyperparathyroidism	01	1:0	2%	0.35%
IgA Nephropathy	01	0:1	2%	0.35%
Fanconi's syndrome	01	0:1	2%	0.35%
Focal segmental glomerulosclerosis	01	0:1	2%	0.35%
Vesico-ureteral reflux disease	01	1:0	2%	0.35%

The above table no. II shows the presentation of various diseases that presented as asymptomatic urinary abnormalities in this study .The male to female ratio has been shown and the prevalence of each such disease has been shown in this table . The above table also shows the contribution which various diseases make to asymptomatic urinary

PRESENTATION OF VARIOUS ASYMPTOMATIC DISEASES

(A)Diabetes Mellitus

Age group	No. of Cases	Presented as
0-12	00	None
13-20	00	None
21-40	06	2 Gly. 2 Pr. 2 Gly.+ py.
41-60	05	2 Gly. 2Py. 1 Pr.
61& above	02	1 Gly.+py. 1 Gly.+pr.

Out of Total 13 cases = 4 presented as Glycosuria(30.76%) ; 3 presented as Proteinuria(23.07%) ; 2 presented as Pyuria(15.38%) ; 3 presented asGlycosuria +Pyuria(23.07%) ; 1 Pesented as Glycosuria +Proteinuria(7.7%). Most common presentation was as glycosuria(30.76%) and least common was as glycosuria + proteinuria(7.7%).

(B) Urinary tract infection

Age group	No of case	Presented as
0-12	00	None
13-20	03	All Py.
21-40	05	All Py.
41-60	03	All Py.
61& above	00	None

Total 11 cases of asymptomatic Urinary tract infection of were detected in this study and all (100%) of them presented as pyuria .

(C) NEPHROLITHIASIS

Age group	No of case	Presented as
0-12	00	None
13-20	00	None
21-40	06	2 ox. 1 ox. +py. 1 ox.+py.+He. 1 He. 1 Py.
41-60	01	Py.+ox.+ph.
61& above	00	None
Total	07	2 Ox. ; 1 Ox.+ Py. 1 Ox+Py.+He 1 Ox.+Py.+Ph. 1 He.

Total 7 cases detected = of which 2 presented as calcium oxalate crystalluria(28.57%) ; 1 as oxalate + pyuria(14.28%) ; 1 as oxalate + pyuria + hematuria(14.28%) ; 1 as oxalate + hematuria + phosphate crystals(14.27%) ; and 1 as isolated hematuria(14.27%).Most common presentation was as asymptomatic calcium oxalate crystals Other presentations were calcium oxalate crystals with other abnormalities like hematuria , pyuria and phosphate crystals .

(D) HTN Nephropathy

Age Group	No of Case	Presented as
0-12	None	-
13-20	None	-
21-40	None	-
41-60	06	5 as pr. 1 as Pr.+ He.
61& above	01	1 as Pr.
Total	07	6 as pr. 1 as Pr. + He.

Total 7 cases detected= 6 presented as proteinuria(85.77%) and 1 presented as proteinuria with hematuria(14.23%). Most common presentation was as proteinuria .

(E) Chronic glomerulo nephritis

Age Group	No of Case	Presented as
21-40	03	All as pr.

Total 3 cases detected and all (100%) presented as proteinuria .

(F) Benign prostatic hypertrophy

Age Group	No of Cases	Presented as
41-60	01	1 as Py.
61 & above	03	1 Py. 1 Py. + Pr. 1Py. + He.
Total	04	2 Py. 1 Py. +pr. 1 Py. + He.

Total 4 cases detected = 2 presented as pyuria (50 %) ; 1 as pyuria + proteinuria (25%) ; and 1 as proteinuria + hematuria (25 %). Most common presentation was as pyuria.

(G) Renal Amyloidosis

Age Group	No of cases	Presented as
41-60	01	Pr.

Only one case seen in this study that presented as proteinuria(100%) .

(H) Hyperparathyroidism

Age Group	No of cases	Presented as
21-40	01	Ox.

Only 1 case of asymptomatic hyperparathyroidism detected that presented as calcium oxalate crysytalluria(100 %)

(I) IgA Nephopathy

Age Group	No of cases	Presented as
0-12	01	Pr.+He.

Only 1 case seen that presented as proteinuria with hematuria (100%)

(J) Fanconi's syndrome

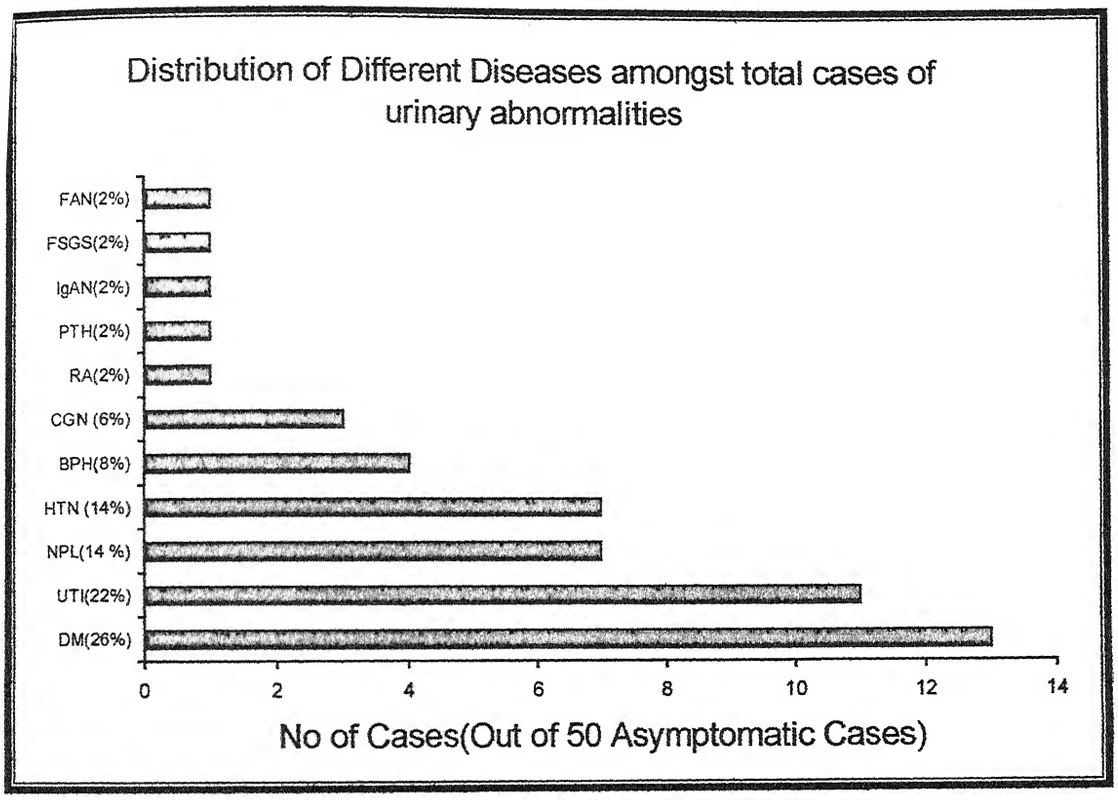
Age Group	No of cases	Presented as
0-12	01	Gly.

Only 1 case seen that presented as Glycosuria (100%).

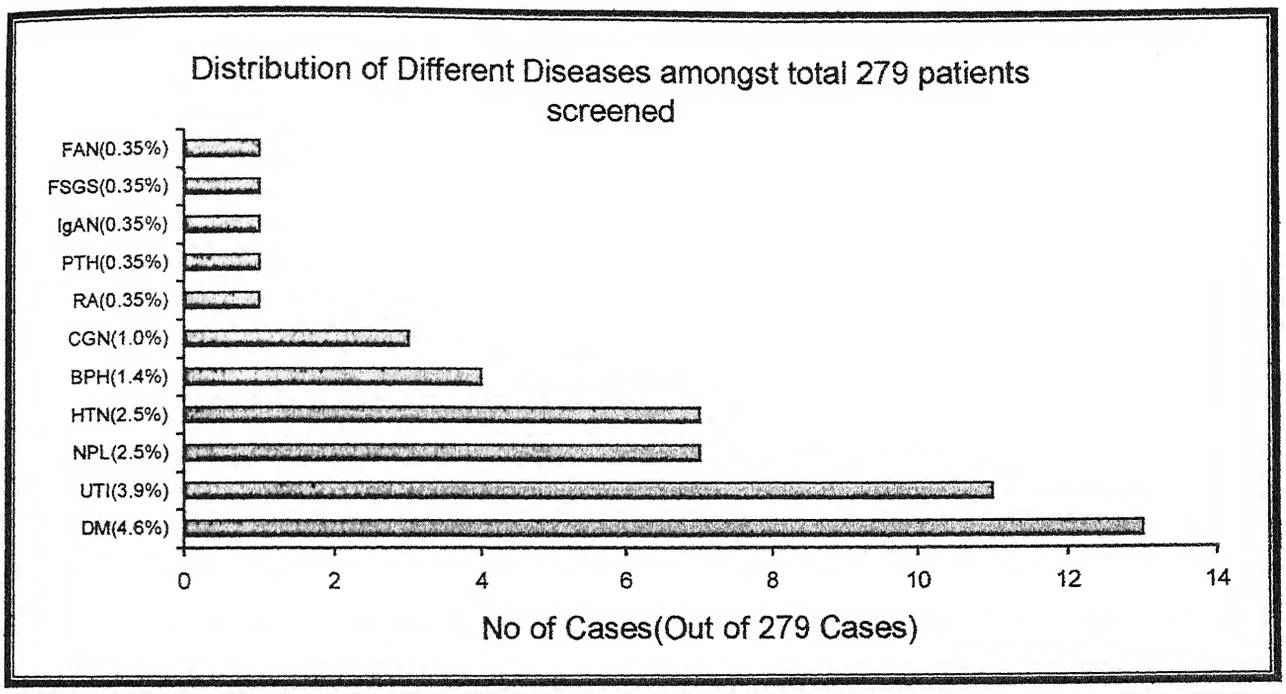
(K) Focal segmental glomerulosclerosis

Age Group	No of cases	Presented as
0-12	01	Pr.

Only 1 case seen that presented as proteinuria (100%) .



As shown in the above table , Diabetes mellitus (DM) was seen as 26% of all asymptomatic urinary abnormalities , Urinary tract infection (UTI) was seen as 22% of all asymptomatic urinary abnormalities, Nephrolithiasis(NPL) was seen as 14% of all asymptomatic urinary abnormalities, Hypertension (HTN) was seen as 14% of all asymptomatic urinary abnormalities, Benign prostatic hyperplasia(BPH) was seen as 8% of all asymptomatic urinary abnormalities , Chronic glomerulonephritis (CGN) was seen as 6% of all asymptomatic urinary abnormalities , Renal amyliodosis (RA) as 2% , Hyperparathyroidism (PTH) as 2% , IgA Nephropathy(IgA N) as 2% , Focal segmental glomerulosclerosis (FSGS) as 2% and Fanconi's syndrome (FAN) as 2% of all asymptomatic urinary abnormalities.



As shown in the above table , Diabetes mellitus (DM) was seen in 4.6% all people screened , Urinary tract infection (UTI) was seen in 3.9% of all people screened, Nephrolithiasis(NPL) was seen in 2.5% of all people screened, Hypertension (HTN) was seen in 2.5% of all people screened, Benign prostatic hyperplasia(BPH) was seen in 1.4% of all people screened , Chronic glomerulonephritis(CGN) was seen in 1% of all people screened, Renal amyliodosis(RA) in 0.35% , Hyperparathyroidism (PTH) in 0.35% , IgA Nephropathy(IgA N) in 0.35% , Focal segmental glomerulosclerosis (FSGS) in 0.35% and Fanconi's syndrome (FAN) in 0.35% of all people screened.

DISCUSSION

DISCUSSION

This study was conducted in Bundelkhand region with the help of renal disease detection camps. In these camps, all age and sex groups patients attending the renal camps were screened by urine routine and microscopic examination.

A total of 279 patients of various age and sex groups were screened by urine examination .Of these 279 cases, 172 were males and 107 were females making a male to female ratio of 1.6:1. For convenience of screening, the study population was divided into discrete study groups according to age. The maximum number of patients who attended the renal camps belonged to the age group 21-40 years and minimum to 61and above years group.

A very similar study has been done by N. Vidya Acharya et al. This study conducted in the city of Bombay involved the study of 430 subjects. A similar division according to age was also done in this study. In addition in this study done by N. Vidya acharya et al, the subjects attending the renal camps were divided further into low socio economic group and middle socio economic group. In the present study in Bundelkhand region however, no such division could be been done, because almost all patients who attended our renal camps belonged to the low economic group thus the scope for comparison was minimal.

In the study asymptomatic urinary abnormalities in Bundelkhand region, out of 279 patients screened by urine routine microscopy examination 68 patients turned out to have detectable urinary abnormality

in their urine. These included symptomatic cases, follow up cases and fresh asymptomatic cases. These 68 cases out of 279 made up 24.37%. In the study of N. Vidya acharya et al , out of 430 patients screened , 151 had detectable urinary abnormalities , this made up 45.5% of the study population . Thus as compared to this 45% of detectable urinary abnormality in the Bombay study, the present study here in Bundelkhand region had 24.37% of detectable urinary abnormality. These 24.37% cases of urinary abnormality included proteinuria, hematuria, Pyuria, crystalluria and Glycosuria. In the study conducted in Bombay however, the only urinary abnormalities stressed on were hematuria, proteinuria and ketonuria, while pyuria and crystalluria were not screened for. In the study done by N.Vidya Acharya et al, the method used to detect asymptomatic urinary abnormalities was dipsticks examination of urine, while method used the present study here was routine and microscopic examination of urine.

Amongst the 68 patients out of 279 detected to have urinary abnormalities, 50 (17.9%) cases were asymptomatic .The maximum prevalence of patients with asymptomatic urinary abnormalities was seen in the age group 61 and above (30%) and the minimum prevalence in 13-20 years age group (11.11%). In this study of 279 people 143 were males and out of these 29 had asymptomatic urinary abnormalities (20.27%), while 86 were females out of which 21 were having asymptomatic urinary abnormalities (24.11%). Thus females were found to have asymptomatic urinary abnormalities a little more frequently as compared to males. In males asymptomatic urinary abnormalities were most prevalent in the age group 61 and above but maximum cases were seen in 21-40 years age group, while in females it was most prevalent in the group 41-60 years with maximum number of cases in 21-40 years

group . Minimum prevalence was seen in 13-20 years age group in both sexes.

Amongst various asymptomatic urinary abnormalities , asymptomatic proteinuria was found in 19 cases out of 50 cases of asymptomatic urinary abnormalities thus making 38 % of this group (19 out of 50). Out of these 19 patients, 14 had isolated proteinuria making 28% asymptomatic urinary abnormalities (14 out of 50). Considering these 19 cases of proteinuria and 14 cases of isolated proteinuria as a percentage of the whole population screened (279 people) , proteinuria was seen in 6.9% (19 out of 279) and isolated proteinuria in 5% (14 out of 279) . In the pediatric age group 0-12 years number of cases of proteinuria were 2 and number of total people screened in this age group were 25 ,thus the prevalence of proteinuria was 8% (2 out of 25) in this study as compared to 4% in the study done by Pygia M.J. , Lott JA et al who studied 6197 school children in 1974 in Japan .In the study done by N. Vidya acharya et al , the prevalence of proteinuria was 23.2% in the whole study group and of isolated proteinuria was 7.2% . Amongst the causes of asymptomatic proteinuria, Hypertension was seen in 42.1% , Diabetes material in 21.2% ,Chronic glomerulo nephritis in 15.8% , Renal Amyloidosis in 5.2% , Benign prostatic hyperplasia in 5.2% , Focal segmental glomerulosclerosis in 5.2% and IgA Nephropathy in 5.2% cases of Asymptomatic proteinuria.

Asymptomatic Pyuria was detected in 26 patients out of 50 patients with asymptomatic abnormality making 52% (26 out of 50). Out of there 26 patients, 17 had isolated pyuria making 34% (14 out of 50). Maximum number of Pyuria in both sexes was seen in the age group 21-40 years (sexually active group). Amongst all the causes of Asymptomatic pyuria, urinary tract infection was seen in 42.3% (11 out of 26) , Diabetes mellitus

in 23.1% (6 out of 26) , Nephrolithiasis in 15.3% (4 out of 26) , Benign prostatic hyperplasia in 15.3% (4 out of 20) and Vesico ureteral reflux in 3.8% (1 out of 26) . In the study of N.V. Acharya et al, pyuria was not screened for and nor has it been screened in other studies, so a comparison could not be made as to what prevalence of asymptomatic pyuria is there in other parts of the country or world.

Asymptomatic Glycosuria was detected in 8 cases out of all patients (50) of asymptomatic urinary abnormabilities , thus it was 16% (5 out of 50) while isolated glycosuria made up 8%(4 out of 50)of all asymptomatic urinary abnormalities . Majority of cases detected were in age group 21-40 and 41-60 years but maximum prevalence was for the age group 61 and above. Only 1 case was seen in 0-12 years (pediatric) age, and the cause was Fanconi's syndrome. In all other cases, the cause of Glycosuria turned out to be Diabetes mellitus. Therefore causes of glycosuria were, Diabetes mellitus 87.5% (7 out of 8) and Fanconi 's syndrome 12.5% (1 out of 8) .Glycosuria when considered as a fraction of the whole population screened , was found to be 2.86% (8 out of 279) . The study of V.N. Acharya et al showed a prevalence of glycosuria in 4.4%.

Asymptomatic Crystalluria was detected in 6 cases out of 50 cases, thus prevalence was 12% (6 out of 50) .Maximum number of cases were detected in the age group 21-40 years and was exclusively detected in male population in this study. Out of all cases asymptomatic crytalluria detected (6) majority were due to Nephrolithiasis 83.3% (5 out of 6) and another less frequent cause was Hyperparathyroidism 16.7%(1 out of 6).

Amongst all cases of Asymptomatic urinary abnormalities (50), hematuria was seen in 5 cases thus making 10% (5 out of 50) and isolated

hematuria was seen in 1 case 2% (1 out of 50). Maximum number of cases of hematuria were seen in the 21-40 years age group (3 cases), but the prevalence was most in pediatric age group (25% of all asymptomatic urinary abnormalities. Considering it as a percentage of total population screened the prevalence was 1.79 %(5 out of 279). In the pediatric age group the prevalence was 8% (1 out of 25 cases). In the study by Vidya N Acharya et al, the prevalence of isolated proteinuria was found out to be 3.9% and that of asymptomatic hematuria with proteinuria was found out to be 16.6% . Amongst the causes of Asymptomatic hematuria , Nephrolithiasis was seen in 40 % (2 out of 5) , while IgA Nephropathy , Hypertensive nephropathy , and Benign prostatic hyperplasia made 20% each (1 out 5 cases each) .

Discussing all Asymptomatic urinary abnormalities together, it was seen that , out of 50 cases of Asymptomatic urinary abnormalities 26 had Asymptomatic pyuria thus making 52% of all Asymptomatic urinary abnormalities , 19 had asymptomatic proteinuria making 38% of all Asymptomatic urinary abnormalities , 8 had asymptomatic glycosuria thus making 16% of all Asymptomatic urinary abnormalities , 6 had asymptomatic crystalluria thus making 12% of all Asymptomatic urinary abnormalities , 5 had asymptomatic hematuria making 10 % of all Asymptomatic urinary abnormalities.

Considering each type of Asymptomatic urinary abnormalities as a percentage of the whole population screened , pyuria was found in 9.3% of all cases (26 out of 279) , proteinuria in 6.8% (19 out of 279) , glycosuria in 2.8% (8 out of 279) , crystalluria in 2.15% (6 out of 279) and hematuria in 1.79% (5 out of 279). A variety of diseases were detected as causes of

asymptomatic urinary abnormalities in this study the details of which has been discussed as below.

Of all diseases, there were 13 cases of Diabetes mellitus of which 6 were males and 7 were females. Thus out of 50 cases of asymptomatic cases detected 13 were due to diabetes making 26 %(13 out of 50). Considering Diabetes in context to the whole study group, it made up 4.65 %(13 out of 279). Out of Total 13 cases 4 presented as Glycosuria(30.76%) ; 3 presented as Proteinuria(23.07%) ; 2 presented as Pyuria(15.38%) ; 3 presented as Glycosuria +Pyuria(23.07%) ; 1 Presented as Glycosuria +Proteinuria(7.7%). Most common presentation was as glycosuria(30.76%) and least common was as glycosuria + proteinuria(7.7%).

Another commonly detected disease as cause of asymptomatic urinary abnormalities was asymptomatic urinary tract infection .There were total 11 cases of asymptomatic Urinary tract infection detected. Out of these 2 were males and 9 were females. Thus Urinary tract infection was 22% of all asymptomatic urinary abnormalities (11 out of 50 cases). Considering asymptomatic Urinary tract infection in the whole study population, it made up 3.94 %(22 out of 279 cases). Total 11 cases of asymptomatic Urinary tract infection of were detected in this study and all (100%) of them presented as pyuria.

There were 7 cases of asymptomatic Nephrolithiasis detected in this study. Out of these all of them were males. Thus asymptomatic Nephrolithiasis made up 14% (7 out of 50 cases) of asymptomatic urinary abnormalities. Nephrolithiasis was seen in 2.51% of total people screened (7 out of 279) .Total 7 cases of Nephrolithiasis were detected , of which 2

presented as calcium oxalate crystalluria(28.57%) ; 1 as oxalate crystals + pyuria (14.28%) ; 1 as oxalate crystals + pyuria + hematuria(14.28%) ; 1 as oxalate crystals + hematuria + phosphate crystals(14.27%) ; and 1 as isolated hematuria (14.27%) .Most common presentation was as asymptomatic calcium oxalate crystals. Other presentations were calcium oxalate crystals with other abnormalities like hematuria, pyuria and phosphate crystals.

Out of 50 cases of asymptomatic abnormalities, Hypertensive nephropathy was detected in 7 cases. Out of these 5 were males and 2 were females. Thus Hypertensive nephropathy made up 14% (7 out of 50) of all asymptomatic urinary abnormalities. Hypertensive nephropathy was seen in 2.51% (7 out of 279) of total patients screened. Of total 7 cases detected, 6 presented as proteinuria (85.77%) and 1 presented as proteinuria with hematuria (14.23%). Most common presentation was as proteinuria.

There were 3 cases of chronic glomerulonephritis seen out of 50 cases thus making 6% (3 of 50) of all asymptomatic urinary abnormalities. All cases were males. It was seen in 1.07% (3 out of 279 cases) of the study population. Total 3 cases detected and all (100%) presented as proteinuria.

There were 4 cases of asymptomatic Benign prostatic hyperplasia making 8% (4 out of 50) of all asymptomatic urinary abnormalities. All these cases were males. Considering it in the total population screened, it was seen in 1.43% of the total study population (4 out of 279). Total 4 cases detected 2 presented as pyuria (50 %) ; 1 as pyuria + proteinuria (

25%) ; and 1 as proteinuria + hematuria (25 %). Most common presentation was as pyuria.

Only 1 case of Renal Amyloidosis was seen, thus making 2% of all asymptomatic urinary abnormalities (1 out of 50). This case was male. Considering the total study population, Renal Amyloidosis was seen in 0.35%(1 out of 279) . This case presented as proteinuria.

Only 1 case of Hyperparathyroidism was seen, thus making 2% of all asymptomatic urinary abnormalities (1 out of 50). This case was male. Considering the total study population, Hyperparathyroidism was seen in 0.35 %(1 out of 279). This case presented as oxalate crysatalluria.
Only 1 case of IgA Nephropathy was seen, thus making 2% of all asymptomatic urinary abnormalities (1 out of 50) .This case was female. Considering the total study population, IgA Nephropathy was seen in 0.35 %(1 out of 279). This case presented as hematuria and proteinuria.

Only 1 case of Fanconi's syndrome was seen, thus making 2% of all asymptomatic urinary abnormalities (1 out of 50). This case was female. Considering the total study population, Fanconi's syndrome was seen in 0.35 %(1 out of 279) . This case presented as glycosuria.

Only 1 case of Focal segmental glomerulosclerosis was seen, thus making 2% of all asymptomatic urinary abnormalities (1 out of 50). This case was female. Considering the total study population, Focal segmental glomerulosclerosis was seen in 0.35 %(1 out of 279). This case presented as proteinuria.

Only 1 case of Vesicuo ureteral reflux disease was seen, thus making 2% of all asymptomatic urinary abnormalities (1 out of 50). This case was female. Considering the total study population Vesicuo ureteral reflux disease was seen in 0.35 %(1 out of 279). This case presented as pyuria.

SUMMERY & CONCLUSION

SUMMARY & CONCLUSION

To summarize, this study which was conducted in Maharani Laxmi Bai medical college, Jhansi with the help of frequently organized organized Renal diseases detection camps in various regions of Bundelkhand region, found out that :

1. Asymptomatic urinary abnormalities were not as infrequent as is generally thought.

2. Of the total 279 patients screened by urine routine microscopy in renal camps, Asymptomatic urinary abnormalities were detected in 17.9% people, who never knew or had any symptoms of renal disease .

3. Various Asymptomatic urinary abnormalities detected in this study were Proteinuria, Pyuria , Hematuria , Crystalluria and Glycosuria .

4. Most commonly prevalent Asymptomatic urinary abnormality in this study was Asymptomatic Pyuria .It was found in 9.3% cases screened. Most common detected cause of asymptomatic pyuria was urinary tract infection. Others causes were Diabetes Mellitus, Nephrolithiasis , BPH and Vesico ureteral reflux disease(uncommon).

5. Asymptomatic Proteinuria was found in 6.8% people screened. Isolated proteinuria was found in 5% of population. Most common cause of proteinuria was Hypertensive nephropathy. Other common causes of

Asymptomatic proteinuria were Diabetes Mellitus and Chronic glomerulo nephritis.

6. Asymptomatic Glycosuria was detected in 2.9% of the population screened . Most common cause of asymptomatic Glycosuria was Diabetes Mellitus with an almost equal distribution in both sexes .

7. Asymptomatic Crystalluria was detected in 2.1% of population screened. It was found exclusively in male population and was most commonly associated with Nephrolithiasis.

8. Asymptomatic Hematuria was detected in 1.7% of the total patients screened while isolated Hematuria was detected in 0.35%. Most common cause of Asymptomatic Hematuria in this study was Nephrolithiasis (found as cause in 40% cases of asymptomatic hematuria detected). While IgA Nephropathy Hypertensive nephropathy and Benign prostatic hyperplasia were other causes.

9. During follow up study of cases detected to have asymptomatic urinary abnormalities, various modalities of investigations were used to reach a conclusive diagnosis for each case . A variety of diseases were detected as cause of these asymptomatic urinary abnormalities , which included Diabets Mellitus, Hypertension , Urinary tract infection , Nephrolithiasis, Benign prostatic hyperplasia , Chronic glomerulonephritis, Renal Amyloidosis , Hyperparathyroidism , IgA Nephropathy , Focal segmental glomerulosclerosis and Fanconi's syndrome.

10. Asymptomatic Diabetes Mellitus was most commonly detected cause of Asymptomatic urinary abnormalities in this study contributing 26% of all Asymptomatic urinary abnormalities detected. Asymptomatic Diabetes mellitus was detected in 4.6% of the population screened.

11. Asymptomatic urinary tract infection was not uncommon and was detected in 3.94% of all cases screened with a male: female ratio of 2:9.

12. Nephrolithiasis and Hypertensive Nephropathy were each detected in 2.5% of total population screened with a male predominance in both diseases.

13. Chronic Glomerulonephritis and Benign Prostatic hyperplasia were detected in 1.07% and 1.43% of the population screened respectively. Both of there diseases showed male predominance .

14. Renal Amyloidosis, Hyperparathyroidism, IgA Nephropathy. Fanconi's Syndrome , Focal segmental Glomerulosclerosis and vesciculo ureteral reflux were other diseases detected in this study as causes of Asymptomatic urinary abnormalities each detected in 0.35% of the total population screened.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Alex M. Davidson and Jean Pierre Grunfeld History and clinical examination of patients with reanal disease (1998) Page 3,4,5,6.
2. Macgvgav G.A. Taskar, P.R.W. & De Wardena H..E. In Disease of the kideney (4th Ed.) pp 2743-53.
3. Burden R.P. Booth L.J. Ockendan, B.G. ryod W.N. Higgius P.Mck(975) Vascular changes in adult patients with recurrent and Loin pain.
4. Karl Shorechi Jacob green, Banj M. Brenner (Chronic Rond failure) Hamrori's 15th Ed Pg. 1552-53.
5. Idiopathic masangial deposite of IgA. In Hamberger J. Crosnier J. and Gwnfeild J.P. Nephrology (1979) p.535.
6. Birch D.F. Fairley , K.F. Whitworth J.A. et al urinary erthcocyte morphology in diagnosis of glomerula hemsturia. Clin. Nephrology 20 : 78 1983.
7. Robert M. Benner Bany H. Branne Disorder of the kidney and urinary Tract Harrison 15th Ed Pg. 1535.
8. Hugh R. Brandy, Yronne N. O Meara Barry M Brenner (Major Glomerulo pathjes) Harrison Ed- 15 Pg 1588-90.
9. HAAS M et al ; changing etiologies of unexplained Nephrotic syndrome; Am J Kidney dis 30 : 621 ; 1997
10. Orth SR RITZ E : The nephritic syndrome N. Engl J Med 338 1202 1998
11. Shu KH, Ho WL, Lu YS, Cheng CH, Wu MJ, Lian JD Long –term outcome of adult patients with minimal urinary abnormalities and normal renal function. Clin Nephrol 1999 Jul; 52(1): 5-9
12. Monhart V, Marek J, Krnch O Ustredni vojenska nemocnice, Praha Isolated hematuria in adolescents and adults. The value of renal biopsy Vnitr Lek 1993 Jan; 39(1): 24-30

13. Topham PS, Harper SJ, Furness PN, Harris KP, Walls J, Feehally Vnitr Lek 1993 Jan; 39(1): 24-30 Q J Med 1994 Jun; 87(6): 329-35.
14. Hrvacevic R, Dirnitrijevic J, SpasicP, Butorajac J, Jovanovic D Histopathologic changes in the kidneys in patients with asymptomatic pathologic findings in urine Vojnosanit Pregl 1998 Sep-Oct; 55(5): 477-82
15. Kitagawa T. lession learned from the Japanese nephritis screening study Pediatr . Nephrol 1989;256-563.
16. Murakami m . Yamamoto H . Udey Murkamik Yamaguchi K . Urinary Screening over 1 13 year period in Tokyo pecarr Nephro 1991; 5: 50-53.
17. Vehaskari V Rapola J. Isolated proteinuria; Analysis of School Age population J Peditr; 1982; 101; 661-668.
18. Litaka k Igarashi S; Sakai T . Hypocomplemectaemia & membraboproliferative Glomerulonephritis in School Urinary Screening in Japan, Nephrol 1994; 8:420-422.
19. K. Yamangata .Y. Yamangata , M Kobayashi & Koyama . A long term follo-up study of asymptomatic haematuria &/or protein uria in adults.Clinical nephrology 1996;45:218-288.
20. YamangataK; Tamida C: Kobayashi K : Prevalence of urinary abnormalities in Asymptomatic Workmes , Koyama A and Yamangata Y . In abstract of XIV the International Congress of Nephrology (1722) Nephrology 197,3:502.
21. Glassock R.J. Alder , S.G. ward It J and Cohen . A H. Primary glomerular disease. In Brenner ,B.M. and Rector F.C. Jr (eds) . The kidney. 3rd .W.B. Saunders , Philadelpha ,1986,p 929.
22. Rosen S. Galyank.E Levy M. and Habib . R : Glomerular disease . Hum Pathol 12:964,1981.
23. Cameron, J, S.and Glassok. R .J. The Nephrotic syndrome, Marcel Dekker , New York ,1988.

24. Goldwasser P. Antigani , Norbergs A et al Urinary red cell blood cell volume differentiates glomerular and non –glomerular hematuria . Kidney int 33: 191, 1988.

25. Scichiri M Hosoda K Nishio Y etal ; Red cell volume distribution curves in diagnosis of glomerular and non –glomerular hematuria lancet 1:1908,1988.

26. West C.D. Asymptomatic hematuria and proteinuria in children cause and appropriate diagnostic studies .J. pediatr. 89:173,1976.

27. Sinniah , R Pwce .H.S. and Lim C.H. Glomerular lesion in Asymptomatic microscopic hemutaruia discorved on routine medical examination Clin Nephrol 5:2;6 1976.

28. Helder .E. ; Kashgarian M and Hayslett J: Clinicopathological correlation of primary hematuria . lancet 1:458,1972.

29. Clarkson .A.R.Woodroffe .A. J. Bannister ,K.M. et al : The syndrome of IgA nephropathy Clin Nephrol 21:7,1984.

30. Emancipator .S. and Schena F.P. (ed) : immunoglobulin A nephropathy . Semin Nephrol 7:275,1987.

31. Shigematsu,H Kobayashi Y. Tateno S et al : Ultrastructiral glomerular loop abnormalities in IgA nephritis . Nephron 30:1,1982.

32. D Amico .G : The commonest glomerulnephritis in the world IgA nephropathy . O.J. Med 64:709, 1987.

33. Lofano , L Garcia – Hoyo R and Egido J IgA nephropathy ; association of a history of microscopic hematuria episodes with increased production of polymetric IgA . Nephron 45:98,1987.

34. Praga .M Gutierrez- Millet V , Navas .J.J. et al . Acute worsening of renal function during episodes of macroscopic hematuria in IgA nephropathy and cyclical neutropaenia Aust. N.Z. J Med 8:184, 1978.

35. Geiger . H a study of complment components C3, C5.C6, C7, C8, C9 in chronic membranoproliferative glomerulonephritis , systemic lupus

erythematosus , poststreptococcal nephritis , idiopathic nephrotic syndrome and anaphylactoid purpura Z kinderheilkd 119:269,1975.

36. Whitworth .J.A.; Turner D.A. Leibowitz S. and Cameron .J.S. Focal segmental sclerosis or scarred focal proliferative glomerulonephritis . Clin Nephrol 9:229,1978.
37. Yoshikawa .N Lijima .K Machara, K et al . Mesangial changes in IgA nephropathy in children, Kidney Int. 32:585,1987.
38. Tiebosch A.T.M.G. Fredrik .P.M. van breda Vreisman ,P. et al Thin-basement membrane nephropathy in adults with persistent hematuria N Engl,J Med.320:14.1989.
39. Robison ,R.R. and Krueger,R.P. Postural proteinuria in Edelman, C.M. Jr(ed) : Pediatric kidney Diseases. Little, Brown & Co., Boston.1979.p597.
40. Vehaskari ,V.M. and Rapola,J. Isolated proteinuria Analysis of a school age population .J Pediatrics 101:661,1982.
41. Yoshikawa ,N, Vehara.S. Yamana ,K et al : Clinicopathological correlation of persistent Asymptomatic proteinuria in children Nephron 25:127,1980.
42. Robinson ,R.R. isolated proteinuria in Asymptomatic patients kidney Int,18:395,1980.
43. Smiah,R LawC.H. and Pwee.H.S.Glomercular lesions in patients with Asymptomatic persistent and orthostatic proteinuria discorved on routine medical examination Clin. Nephrol 7:1.1977.
44. Ramirez F . Travis ,L.B. Cunningham,R.J. et al Focal segmental glomerulosclerosis, crescent and rapidly progressive renal failure Int,J.Pediatr. Nephrol,3:175,1982.
45. COUSER WG Glomerulonephritis Lancer 353:1509, 1999.
46. D.AMICO G.Pathogenesis of immunoglobulin A nephropathy. Curr opin Nephrol Hypertens 7:247,1998.

47. D Das M. et al changing etiologies of unexplained adult nephrotic syndrome: A comparison of renal biopsy finding from 1976-1979 and 1995-1997Am J Kidney Dis 30:621,1997.

48. HRICK DE et al GlomerulonephritisN Engl J Med 339:889,1998.

49. BOROHI L et al : Urinary volume water and recurrences in idiopathic calcium nephrolithiasis. A 5 year randomized prospective study .J. Urol 155:839,1996.

50. SEGURA .JW et al Ureteral Stones clinical Guidelines panel summary report on the management of ureteral calculi . J Urol 138:1915,1997.

51. A prospective study of the C, Grave W, de Heer F, de Leeuw P, Zeppenfeldt E, Breda Vriesman PJ.

52. Asymptomatic isolated microhaematuria : natural history of 136 children.

53. Symptomless microhaematuria in schoolchildren: causes for variable management strategies.

54. Guide to clinical preventive services 2nd ed. Williams & Wilkins, Baltimore , 1996;PP181-6.

55. Kasiske BL, Keane WF, Laboratory assessment of renal disease: Clearance urinalysis and renal biopsy in the kidney (6th ed) : Brenner BM,WB Saunders , Philadelphia 2000;pp 1142-53.

56. Norvin Peter AF. Urinary sediment in the interpretation of proteinuria. Annals of internal Medicine 1983; 98:254-5.

57. Schumann GB , Greenberg NF , Usefulness of microscopic urinalysis as a screening procedure . A preliminary report .Am Clin Pathol 1979;71:4 52-6.

58. Cockroft D.W. Gault MH. Pedicition of creatinine clearance from serum creatinine. Nephron. 1976,16:31.

59. De Wardener H.E. (1985) , The excretion of acid in renal disease . Quart J Med . 1959,28,259-313.

60. Lalla MLT, Finnish recommendation for methods used in urine analysis: Results of five years period . Scandinavian J Lab Invest 1990,50,34-40.

61. Schawb SJ, Christensen L, Doughtry K , and Klahr S. Quantification of proteinuria by the used of protein to creatinine ratio in single urine sample Arch Intern Med. 1987,147:943.

62. Ginberg J.M. Change B.S., Matarese , R.A. and Garella , S. Use of single voided urine smples to estimate quantitative proteinuria n Engl.J Med 1983,309:1543.

63. Rath B, Turner C, Hartely B and Chanler C. What makes red cell dysmorphic in glomerular haematuria? Paediatr Naphrol, 1992,6,424-7.

64. Addis T. The number of formed elements in the urinary sediment of normal individual , j Clin invest 1926;2:409.

65. Larcom RC, Carter GH, Erythocytes in urinary sediment identification and normal limits J Lab Clin Med 1948;33:875.

66. Ritchie CD, Bevan EA , Collier SJ , Importance of occult hematuria : found at screening Br Med J 1986;292:681-3.

67. Mohr DN offord KP, Melton L.J. isolated asymptomatic microhematuria :A cross- sectional analysis of test positive and test negative patients. J Gen Intern Med 1987;2:318.

68. Is routine urinalysis worthwhile ? Lancet 1988;1:747.

69. Fairley KF, Birch DF, Hematuria : A simple method for identifying glomerular bleeding kid int. 1982;21:105.

70. Dinda AK Saxena S, Guleraia S et al . Diagnosis of glomerular hematuria: Role of dystrophic red cell GI cell and bright field microscopy. The Scandinavian J of Clinical and Laboratory investigation 1997; 57(3) 203-08.

71. Schichiri M Nishio Y, Suenega M et al . Red cell volume distribution curves in diagnosis of glomerular and non- glomerular hematuria. Lancet 1988; 1:908-11.

72. Offringa M , Benbassat J. The value of urinary red cell shape in the diagnosis of g glomerular and post glomerular hematuria . A meta-analysis. postgrad Med. J 1992;68:648-54.

73. Gyory AZ , Hodfield C, Laner CS. Value of urine microscopy in predicting histological changes in the kidney . Double blind comparison. Br Med J 1984; 288:819-22.

74. Nieuwhof C, Doorenbs C, Grave W, de Heer F, de Leeuw P, Zeppenfeldt E, van Breda Vriesman PJ, A prospective study of the natural history of idiopathic non-proteinuric hematuria Kidney Int 1996 Jan; 49(1): 222-5

75. Hisano S, kwano M, Hatae K, Kaku Y, Yamane I, Ueda K, Uragoh K, Honda S Asymptomatic isolated microhaematuria Hosp Pract (Off Ed) 1996 Jul 15; 31 (7): 106, 109-10

76. Benbassat J, Gergawi M, Offringa M, Drukker A Symptomless microhaematuria in school children QJM 1996 Nov; 89(11):845-54

77. Cho BS, Kim SD, Choi YM, Kang HH. School urinalysis screening in Korea Pediatr Nephrol 2001 Dec;16(12):1126-8

78. Lin CY, Sheng CC, Lin CC, Chen CH, Chou P. Mass urinary screening and follow-up for school children in Taiwan Province. Acta Paediatr Taiwan 2001 May-Jun;42(3):134-40

79. Takebayashi S, Yanase K. Asymptomatic urinary abnormalities found via the Japanese school screening program: a clinical, morphological and prognostic analysis. Nephron 1992;61(1):82-8

80. Wei JN, Chuang LM, Lin CC, Chiang CC, Lin RS, Sung FC Childhood diabetes identified in mass urine screening program in Taiwan, 1993-1999. Diabetes Res Clin Pract 2003 Mar;59(3):201-6

81. Pugia MJ, Murakami M, Lott JA, Ohta Y, Kitagawa T, Yamauchi K, Suhara Y, Kasjima J. Screening for proteinuria in Japanese schoolchildren Clin Chem Lab Med 2000 Oct;38(10):975-82

82. Pugia MJ, Lott JA, Kajima J, Saambe T, Sasaki M, Kuromoto K, Nakamura R, Fusegawa H, Ohta Y Screening school children for albuminuria, proteinuria and occult blood with dipsticks. Clin Chem Lab Med 1999 Feb;37(2):149-57

83. Hiatt RA, Ordonez JD Dipstick urinalysis screening, asymptomatic microhematuria, and subsequent urological cancers in a population-based sample.
Cancer Epidemiol Biomarkers Prev 1994 Sep;3(6):523

84. Plata R, Silva C, Yahuita J, Perez L, Schieppati A, Remuzzi G. Mario Negri The first clinical and epidemiological programme on renal disease in Bolivia: Nephrol Dial Transplant 1998 Dec;13(12):3034-6

85. Britton JP, Dowell AC, Whelan P, Harris CM. A community study of bladder cancer screening by the detection of occult urinary bleeding J Urol 1992 Sep;148(3):788-90

86. Jones CA, Francis ME, Eberhardt MS, Chavers B, Coresh J, Engelgau M, Kusek JW, Byrd-Holt D, Narayan KM, Herman WH, Jones CP, Salive M, Agodoa LY Microalbuminuria in the US population: third National Health and Nutrition Examination Survey ;Am J Kidney Dis 2002 Mar;39(3):445-59.

87. Yamagata K, Takahashi H, Tomida C, Yamagata Y, Koyama A. Prognosis of asymptomatic hematuria and/or proteinuria in men. High prevalence of IgA nephropathy among proteinuric patients found in mass screening. Nephron 2002 May;91(1):34-42

MASTER CHART

MASTER CHART

PART I -RESULTS OF RENAL DISEASE DETECTION CAMPS

Serial No.	Name	Age/Sex	Blood Pressure in mmHg	Clinical features	Urine Routine/microscopy	Blood Sugar	Fundus
1	Smt. Neelima.Shukla	55/F	140/100	Hypothyroidism.	NAD	-95mg%(R)	Not done
2.	Shanti Bai	60/F	. 100/50	FUC/CRF/HTN/ RA 40 Swelling Abdominal pain	Protein ++	72%mg(4/npp)	B/L Cataract
3.	Mehmooda	32/F	110/ 70	FUC. Renal Stone.	NAD	not done	not done
4.	Smt.Sushama Shivhara	32/F	108/76	Burning micturition	Pus cells occasional Rest NAD.	Not done	Not done
5.	Hamida	48/F	100/74	none	NAD	Not done	Not done
6.	Moh Hanif farook	50/M	130/84	none	NAD	Not done	Not done
7.	Km. Aasha	18/F	120/74	Pain in Rt. renal angle 3 days	NAD	Not done	Not done
8.	Ram deen	60/M	144/76	Burning micturition ↑ frequency	NAD	Not done	Not done
9.	Panku	55/M	134/84	Lump in left i.f.	NAD	Not done	Not done
10.	Ramdol	64/M	156/98	none	Sugar++++ Pus cells occasional.	Not done	Not done
11.	Rama malvaya	38/F	126/84	constipation	NAD	Not done	Not done
12.	Vani	50/F	110/70		Pus Cells 3-4/hpf Epith occ.	Not done	Not done
13.	Ghanghey	37/M	120/80	Epigastric pain	Pus cell 1-2	Not done	Not done
14.	Anjana	36/F	110/74	None	Sugar+++ pus cells 12 – 14 /hpf	296mg% 2h pp	Not done
15.	Panveer	55/F	120/70	None	NAD	Not done	Not done
16.	Raj Kumari	38/F	124/80	None	NAD	Not done	Not done
17.	Pram Giri	60/M	110/78	None	NAD	Not done	Not done
18.	Davandra	36/M	108/66	None	NAD	Not done	Not done
19.	Dawarika	49/M	140/80	Left lain pain	NAD	Not done	Not done
20.	Mahd Muskiq	51/M	156/86	Polydypsia polyurea hesitancy	Sugar ++++ Pus cells 6-8 / hpf	Not done	Not done
21.	Smt. Pankuwar	44/F	120/78	none	NAD	Not done	Not done
22.	Ajay Srivastava	40/M	136/90	Known DM	NAD	83mg% 2hrpp	
23.	Maya Devi	22/F	146/90	Back ache	NAD	Not done	Not done
24.	Radharaman.	45/M	110/68	loin pain	Cast hyaline. Rest NAD	Not done	Not done
25.	Shyam Kishor	46/M	124/86	Difficulty micturition	NAD	Not done	Not done
26.	Suresh Chandra	44/M	142/90	↑ frequency Burning mic.	NAD	Not done	Not done
27.	Nagma	31/F	122/70	None	NAD	Not done	Not done

28.	Khilau	30/M	100/60	None	NAD	Not done	Not done
29.	Kusum	35/F	106/80	None	NAD	Not done	Not done
30.	Anjana	36/F	114/68	None	NAD	Not done	Not done
31.	Kirti	10/F	106/60	None	Urine protein ++	Bl. Sugar 94mg%(R)	Not done
32.	L.R. Verma	46/M	102/70	None	Pus cell occasional. Phosphate crystals	Not done	Not done
33.	RamKale	60/M	100/70	Pain in Knee j Ghabrahat	NAD	Not done	Not done
34.	Bhagowerti	40/F	130/80	Arthralgia insomnia Anorexia	Pus cell present	Bl. Sugar 341 mg% (R)	Fundus DR +
35.	Hadid Khan	65/M	160/90	None	Protein++ pus cells 6-8 /hpf	77mg%	Not done
36.	Rana Kaheed	40/F	140/80	None	NAD	Not done	Not done
37.	Mohan	40/M	120/86	None	NAD	Not done	Not done
38.	Nasreem	33/F	126/76	Recurrent UTI	NAD	Not done	Not done
39.	Smt. BD. Singh	53/F	100/70	None	NAD	Not done	Not done
40.	K.P. Singh	62/M	154/90	None	NAD	Not done	Not done
41.	Smt. Kapoori	42/F	180/80	None	NAD	Not done	Not done
42.	Mohd. mushtaque	51/M	140/80	Pain during micturition	Pus cells +++ (Field full)	Bl. Sugar 94mg%	Not done
43.	Lakshmi	42/F	120/76	None	NAD	Not done	Not done
44.	Smt. Sajeeda	45/F	144/86	None	NAD	Not done	Not done
45.	Raj Kumari	35/F	128/80	Known pt. of DM	Protein ++ Sugar Nil	Sugar 154mg% 4hrpp	Not done
46.	Nolha Devi	50/F	110/70	None	NAD	Not done	Not done
47.	Mohan	40/M	100/80	Gen. swelling 5 yr.	NAD	Not done	Not done
48.	Rajan sharma	40/M	146/80	None	NAD	Not done	Not done
49.	Ram dayal	43/M	126/80	None	NAD	Not done	Not done
50.	Salil Bansal	37/M	136/74	None	NAD	Not done	Not done
51.	Smt. Noorjahan	55yr/F	120/80	Burning micturition	Calcium oxalate crystals	Not done	Not done
52.	Smt. Akhtari biwi	45 yr/F	116/72	None	Sugar++++	BL. Sugar 385mg/dl	Not done
53.	Chand Biwi	50/F	118/80	None	Protein traces 5-6 Pus cells/ HPF 2-3 epithelial cells	Not done	Not done
54.	Suttan	8/M	100/76	None	NAD	Not done	Not done
55.	Mr.R.P.Singh	82/M	174/96	None	NAD	Not done	Not done
56.	Avinash	8/M	100/70	None	6-8 pm cells/HPF	Not done	Not done
57.	Sumat Kumar	40/M	106/80	None	NAD	Not done	Not done
58.	Chandra Singh	45 yr/M	110/.80	None	Proteins +	Random Blood sugar 315mg%	Not done
59.	Meenu Shekhar	15 yr/ F	106/70	None	NAD	Not done	Not done
60.	Rajendra Prasad	25/M	126/80	None	NAD	Not done	Not done
61.	Sukhiya	35/F	110/80	None	4-6 per cells/HPF	NAD	Not done
62.	Kamran	26/M	120/80	None	Oxalate crystals seen	NAD	
63.	Gajendra	60 yr/M	164/100	None	Potein, ++	Bl. Sugar(R) 126 mg%	B/L Hazy media
64.	Tahir Husain	12/M	100/70	Occ. Headache + Dimunition of vision	NAD	Not done	Not done
65.	Vidya Devi	28/F	100/76	Mild occasional Headache	NAD	Not done	Not done
66.	Bhagwati Kumar singh	40/F	140/86	None	NAD	Not done	Not done
67.	Vivendra singh	44/M	132/80	Abd dismonful	NAD	Not done	Not done
68.	Jayannath singh	42/M	120/86	Lt. iliac fossa pain	NAD	Not done	Not done
69.	Laxmi shankar	41/M	120/80	None	Traces of albumin present	Not done	Not done
70.	Rampyari	44/F	116/72	None	NAD	Not done	Not done
71.	Ramnarayan singh	42/M	150/78	Abd pain	NAD	Not done	Not done

72.	Vinay kumar	16/M	112/80	palpitation	NAD	Not done	Not done
73.	Rajna	26/F	104/74	None	NAD	Not done	Not done
74.	Fagruddin	51/M	156/80	None	NAD	Not done	Not done
75.	Kunwar behadni	34/M	110/70	nasal blockade + headache	NAD	94mg% (2hrpp)	Not done
76.	Ram Kunwar	35/m	120/78	None	20-30 pus cells/ HPF 15-16 RBC/ hpf ; oxalate crystals present	120mg% (3 hr pp)	Not done
77.	Dhyan Tripathi	31/M	110/80	Headache	NAD	Not done	Not done
78.	Jashods	40/F	140/80	None	NAD	Not done	Not done
79..	Panns lal	40/M	110/90	None	NAD	Not done	Not done
80.	Anil kumar	28/M	120/82	Burning micturition for last 6-7 days	NAD	Bl. Sugar 108mg% (3hrpp)	Not done
81.	Vishram singh	30/M	116/78	Headache	NAD	Not done	Not done
23.	Suresh pratap	41/M	140/80	Epigastric pain	NAD	Not done	Not done
83.	Ram kumar	35/M	120/74	Cough and cold	Sugar+	BL. Sugar = 274mg% (2hrpp)	B/L Normal
84.	Ram Avbaar	26/M	150/104	Headache	Protein ++	Bl. Sugar 98mg%	Papillede ms Lt. eye
85.	Baboo	6/M	Not Recorded	None	NAD	Not done	Not done
86.	Prem Nath	30/M	126/80	None	NAD	Not done	Not done
87.	Naresh Prasad	64/M	105/50	None	6-8 per cells/HPF	Not done	Not done
88.	Preetaim sinth	50/M	120/50	None	NAD	Not done	Not done
89.	Sukhi	32/M	110/70	None	NAD	Not done	Not done
90.	Amit sadan	15/M	120/76	None	NAD	Not done	Not done
91.	Radhika Srivastava	23/F	110/70	Low grade fever	NAD	Not done	Not done
92.	Anbika srivastava	43/F	160/100	None	NAD	Not done	Not done
93.	Kasim Iqbaal	35/M	120/80	8-10 per cells/HPF oxalate, phosphate crystals	NAD	Not done	Not done
94.	Palayanutkal	45/M	130/50	None	NAD	Not done	Not done
95.	Mahesh pandey	40/m	140/50	None	NAD	Not done	Not done
96.	Gudiya	10/F	110/70	None	NAD	Not done	Not done
97.	Shayama	15/F	104/70	Lump in breast	NAD	Not done	Rett to sugar
98.	Ganesh kutums	30/M	120/76	None	NAD	Not done	Not done
99.	Pooran dayal	25/M	120/82	None	NAD	Not done	Not done
100.	Ghan shayam	35/M	130/76	None	NAD	Not done	Not done
101.	Rohit	5/M	140/70	None	NAD	Not done	Not done
102.	Peelu Bhagat	40/M	146/100	obesity	Sugar + protein +	350 mg % 3 hr pp	Not done
103.	Pyaare lal	32/M	120/50	None	NAD	Not done	Not done
104.	Madan Kativaav	30/M	117/76	None	NAD	Not done	Not done
105.	Suneel jain	25/M	120/74	None	NAD	Not done	Not done
106.	Preeti jain	20/F	110/70	None	NAD	Not done	Not done
107.	Anant jain	50/M	110/50	None	NAD	Not done	Not done
108.	Namita Das	23/F	120/76	None	NAD	Not done	Not done
109.	Shobha Das	15/F	100/90	Occasional burning micturition	Pus cells 10-12/HPF Protein +	Not done	Not done
110.	Gaman Das	30/M	110/74	None	NAD	Not done	Not done
111.	Eshwar Gupta	26/M	130/76	None	NAD	Not done	Not done
112.	Tarun purohit	43/M	140/90	Occ. Abd pain	NAD	Not done	Not done
113.	Bheem kumar	25/M	120/70	Scrotal pruritis	NAD	Not done	Not done
114.	Sheela kumar	22/F	100/70	None	NAD	Not done	Not done
115.	Taranum Khan	30/F	140/70	Pregnancy (24 weeks) asymptomatic	Pus cells 6-5/HPF	Not done	Not done
116.	EhsanKhan	36/M	110/90	None	NAD	Not done	Not done

117.	Nidhi pathak	10/F	100/74	Burning micturition	Pus cells 14-16/HPF Protein ++	97 mg % (2 hr pp)	Not done
118.	Rudra Sen	25/M	120/70	None	NAD	Not done	Not done
119.	Rambha chaudhary	45/F	200/130	None	Protein +	294 mg% (4 hr pp)	Not done
120.	Gaya Deen	30/M	140/90	None	NAD	Not done	Not done
121.	Jugal	38/M	120/50	None	NAD	Not done	Not done
122.	Shekhar pal	20/M	110/76	None	NAD	Not done	Not done
123.	Neetu	5/F	150/90	None	Protein++ Red cells = 10-14/HPF	Not done	Not done
124.	Daman	5/M	100/74	None	NAD	Not done	Not done
125.	Ramavva	24/M	110/50	Headache	NAD	Not done	Not done
126.	Chandan	26/M	120/50	Cough + loss of weight (1 month)	NAD	Not done	Not done
127.	Kalku Prasad	50/M	130/86	Chronic cough with expectoration(smoker)	NAD	Not done	Not done
128.	Gomti Devi	32/F	100/76	None	NAD	Not done	Not done
129.	Sudha Devi	36/F	120/78	↑ bleeding during periods	NAD	Not done	Not done
130.	Katori Devi	42/F	104/76	None	NAD	Not done	Not done
131.	Digam babu	33/M	130/82	None	NAD	Not done	Not done
132.	Alatas Khan	40/M	145/94"	Obesity	Sugar + + Pus cells 8-10/HPF	294mg% (3 hr pp)	Not done
133.	Kumari Suneeta	17/F	110/70	None	NAD	Not done	Not done
134.	Jeevan lal	25/M	140/100	None	NAD	Not done	Not done
135.	Gaurav lal	30/M	160/60	None	NAD	Not done	Not done
136.	Prem bai	44/F	120/68	None	NAD	Not done	Not done
137.	Satm Singh	37/M	130/90	None	NAD	Not done	Not done
138.	Sumi Shukla	26/F		None	NAD	Not done	Not done
139.	Prakash Shukla	30/M		None	Phosphate & urate crystals + RBC = 10-15/hpf	Not done	Not done
140.	Chanchal shukla	5/F	140/100	None	Protien ++	78 mg % (4 hr pp)	Not done
141.	Sagun bai	30/F		None	NAD	Not done	Not done
142.	Triveni sood	32/F		None	NAD	Not done	Not done
143.	Arjun Singh	20/M		None	Pus cells 10-15 /HPF	121 mg % (3 hr pp)	Not done
144	Nakul singh	35/M		None	NAD	Not done	Not done
145.	Shobha Ram	48/M	120/50	Fever 2-3 days with cough	NAD	Not done	Not done
146.	Tasar chaube	27/M	126/70	None	NAD	Not done	Not done
147	Shivani Shukla	32/F	136/94	None	NAD	Not done	Not done
148.	Brij kishore Rai	35/M	160/110	None	Protien++	Bl. Gluc 92mg% 4hrpp	Diadetic retinopathy
149.	Umar Mukhtar	42/M	144/94	None	NAD (only occasioned pus cells seen)	Bl. Glucose 254mg%	Diabetes Retiniply
150.	Pyaare Mohan	32/M	124/76	None	Oxalate and phosphate crystals	Not done	Not done
151.	Dilip Dhavia	28/M	130/90	None	NAD	Not done	Not done
152.	Upendra Baijal	32/M	120/50	None	NAD	Not done	Not done
153.	Kahar singh	24/M	190/110	None	Protein=+	140 mg % (2 hr pp)	B/L WNL
154.	Doli Mehta	16/F	105/70	Itching all over body	NAD	Not done	Not done
155.	Lagan Gupta	58/M	149/90	Poor stream formation during urination	Pus cells 10-12/HPF	136mg% (3 ½ hr pp)	Not done
156.	Anand puri	64/M	150/100	None	NAD	Not done	Not done
157.	Shamshad Ansari	45/M	120/80	Itching around the anus	NAD	Not done	Not done
158.	Anwari begum	35/F	116/70	Weekness+ pain in limbs	NAD	Not done	Not done

159.	Pista Devi	40/F	100/70	None	NAD	Not done	Not done
160.	Prabhu bhagat	16/M	118/80	None	NAD	Not done	Not done
161.	Peelu Singh	44/m	134/80	None	Protein =+++	Not done	Not done
162.	Deewakar purohit	50/M	120/50	None	None	Not done	Not done
163.	Mandeep chadha	45/m	150/100	Burning micturition	Pus cells 20-25/HPF Protein ++	314mg%	Diabetic retinopat y seen
164.	Pramesh Tiwari	30?M	124/78	None	NAD	Not done	Not done
165.	Shivangi Misra	34/F	110/70	None	NAD	Not done	Not done
166.	Bahusali	40/F	138/74	None	NAD	Not done	Not done
167.	Preetam Srivastava	22/M	198/120	None	Protein ++	100 mg % (F)	B/L Vessel changes no papillede ma.
168.	GauravAtareja	30/M	126/82	None	NAD	Not done	Not done
169.	Rajan Gupta	32/M	110/70	None	NAD	Not done	Not done
170.	Sugoto Mukherjee	25/M	126/84	None	NAD	Not done	Not done
171.	Deewan Seth	58/M	160/100	None	Protein=++ Pus cells =10-12/HPF	100mg%	could not be seen B/L cataract
172.	Prem Das	70/M	140/96	Known diabetic on drugs	Protein=++	212mg%	B/L cataract
173.	Nimal Saxena	34/M	126/70	None	NAD	Not done	Not done
174.	Gopal Agarwal	15/M	110/70	None	NAD	Not done	Not done
175.	Bheemsen	34/M	124/88	None	Plenty of calcium & oxalate crystals	Not done	Not done
176.	Gonda Ram	12/m	104/100	None	None	Not done	Not done
177.	Madhuri Gupta	5/F	110/70	None	Sugar +++	250mg% (2 hr pp)	B/l Normal
178.	Nitesh Kumar	5/M	100/70	None	NAD	Not done	Not done
179.	Sanwal Pavihaar	35/M	174/100	None	NAD	Not done	Not done
180.	Kastoori Devi	23/F	110/76	None	NAD	Not done	Not done
181.	Urmila sethi	44/F	130/84	None	Sugar ++ Pus cells 15-20/hpf	340mg% (2hrpp)	Not done
182.	Videshi prakash	70/M	120/84	None	NAD	Not done	Not done
183.	Nimnum Shukla	80/M	160/90	None	NAD	138mg% (3hrpp)	Not done
184.	Susheel chaturvedi	24/m	120/78	None	NAD	Not done	Not done
185.	Uttam gorda	60/M	120/50	None	NAD	Not done	Not done
186.	Surrabh singh	74/M	140/50	None	NAD	Bl. Sugar 150mg%	Not done
187.	Mahesh pandey	25/M	126/90	None	NAD	Not done	Not done
188.	Sachin Agarwal	50/M	130/70	None	NAD	Not done	Not done
189.	Pardhan Kumar	74/m	130/94	None	NAD	94mg% (1hrpp)	Hyperten sive vessel changes
190.	Mithu	6/F	154/100	None	NAD	100mg%(R)	B/L normal
191.	Gauri Devi	30/F	110/70	None	NAD	Not done	Not done
192.	Susheela Devi	50/F	100/76	None	Pus cells 12-14/HPF	112mg%(R)	None
193.	Samreth Gupta	20/M	120/56	None	NAD	Not done	Not done
194.	Nisbukl singh	16/M	126/50	None	NAD	Not done	Not done
195.	Azam beg	42/M	10/50	None	NAD	Not done	Not done
196.	Kamini Begum	38/F	150/100	None	Protein=++	349mg%(R)	Diabetic Retinopat hy
197.	Ayaz beg	14/M	120/50	None	NAD	Not done	Not done
198.	Lomvati	25F	105/70	None	NAD	Not done	Not done

199.	Sadhna sethi	24/F	110/50	None	NAD	Not done	Not done
200.	Ramayya Devi	30/F	104/70	Burning micturition	Pus cells 10-12/HPF Protein +	100mg%(R)	Not done
201.	Samved misra	24/M	124/80	None	NAD	Not done	Not done
202.	Rajini misra	60/F	150/100	None	NAD	94 mg %	Not done
203	Ayur kumar	56/M	130/84	None	NAD	Not done	Not done
204	Malkhan	44/M	140/96	None	NAD	Not done	Not done
205	Ninhai kumar	32/M	140/80	None	NAD	Not done	Not done
206.	Dhaniram	26/M	114/50	None	NAD	Not done	Not done
207.	Vishwanath Das	54/M	110/70	Poor Stream formation	Protein (+)	109mg%(R)	Not done
208.	Gajendra singh	64/M	110/70	None	NAD	Not done	Not done
209.	Chatur singh	20/M	108/76	None	NAD	Not done	Not done
210.	Meera Devi	68/F	120/74	None	Sugar ++ Protein ++ Pus cells 10-12/HPF	274mg%(R)	B/L Cataract
211.	Babu lal	34/M	110/70	None	NAD	Not done	Not done
212.	Magbool Khan	36/M	124/50	None	NAD	Not done	Not done
213.	Mashkool beg	72/M	150/100	Poor urinary stream	NAD	108mg% (2 hr pp)	Not done
214.	Sajid Husain	9/M	160/100	Swelling around eyes	Protein ++	97mg% (3 hr pp)	hypertensive changes
215.	Dharmendra Kumar	12/M	150/98	None	NAD	Not done	Not done
216.	Sheela Devi	22/F	100/70	Occasional burning micturition	NAD	Not done	Not done
217.	Ladkunwar	65/F	110/70	Burning micturition + prolapse	Protein + Pus Cells 14-16/hpf	112mg%(R)	Not done
218.	Mankunwar	58/F	210/120	Occasional headaches	Protein=++	114 mg % (R)	B/L Cataract
219.	Altaaf Raza	36/m	124/50	None	NAD	Not done	Not done
220.	Gaman Tyagi	16/M	120/50	None	NAD	Not done	Not done
221.	Sumit sood	22/M	110/74	None	NAD	Not done	Not done
222.	Pyaari	24/F	104/70	None	Pus cells 8-10/HPF	94mg%	Not done
223.	Gurjani Devi	42/F	110/70	None	Pus cells 16-18/HPF	174mg% (F)	Not done
224.	SringarDevi	30/F	132/50	None	NAD	Not done	Not done
225.	Gita Devi	16/F	110/76	None	NAD	Not done	Not done
226.	Naseeb ullah.	39/M	120/84	None	NAD	Not done	Not done
227.	Suman	5/F	Not.Rec.	None	NAD	Not done	Not done
228.	Dhaniram	50/M	120/54	None	NAD	Not done	Not done
229.	Indu Gupta	16/F	110/70	None	NAD	Not done	Not done
230.	Ramdas	74/M	150/104	None	NAD	84mg%	B/L Cataract
231.	Sanjay Tiwari	24/M	120/50	None	NAD	Not done	Not done
232.	Kamla Tiwari	20/F	104/70	None	NAD	Not done	Not done
233.	TriveriGupta	5/F	140/90	None	NAD	110mg%(R)	Fundus Normal
234.	Naman Gupta	34/M	120/50	None	NAD	Not done	Not done
235.	Hardas Singh	727M	110/50	Poor stram of urine+taken long time to pass urine	NAD	BL Sugar 124mg%(R)	B/L Cataract
236.	Guddi	6/F	100/70	None	NAD	Not done	Not done
237.	Purnima	5/F	110/80	None	10-12 pus cells/HPF	154mg%(R)	
238.	Bhagwan das	47/M	1201/56	None	NAD	Not done	Not done
239.	Baby singh	2/F	No recorded	None	NAD	Not done	Not done
240.	Malkan singh	30/M	120/82	None	10-12 Pus cells/HPF	132mg%(R)	
241	Sevaknath	14/M	120/54	None	NAD	Not done	Not done
242.	Dasnishkamal	25/M	124/76	None	NAD	Not done	Not done

243.	Premvati	32/F	110/70	Occ. Renal angle pain	Pus cells 6-8/HPF oxalate + phosphate crystals	Not done	Not done
244.	Deewan singh	40/M	110/50	None	NAD	Not done	Not done
245.	RaniDevi	33/F	120/50	None	NAD	Not done	Not done
246.	Rouma singh	16/F	100/72	None	10-12Pus cells/HPF	Not done	Not done
247.	Subedarsingh	436/M	120/5/8	None	NAD	Not done	Not done
248.	Kiran kumari	5/F	110/70	None	NAD	Not done	Not done
249.	Ganga Devi	66/F	178/98	None	NAD	96mg%(R)	B/L Hazy media
250.	Om Dev	59/M	150/54	Known diabetic (on drugs)	Protein +	Bl. Sugar =166	B/L Cataract
251.	Sripal singh	45/M	120/90	None	NAD	Not done	Not done
252.	Allen Dsouza	55/M	188/104	None	Pus cells 6-8/hpf protein +	104mg%(R)	Hyprtensive vessel changes
253.	Kareem	16/M	110/80	None	NAD	Not done	Not done
254.	Barkat	45/M	110/54	None	Pus cells=10-12/HPF	160mg%(R)	Not done
255.	Laadli	8/F	120/80	None	NAD	75mg% (5hrpp)	Not done
256.	Aroop kundu	35/M	126/70	None	NAD	Not done	Not done
257.	Sadhna kumari	3/F	Not recorded	None	NAD	97mg%(R)	Not done
258.	Bilqees Bano	32/F	110/70	None	NAD	Not done	Not done
259.	Shanaaz Bano	16/F	100/70	None	NAD	Not done	Not done
260.	Rahman Ullah	50/M	150/96	Inabiliy to fall asleep	Pus cells=8-10/HPF	Bl. Sugar =84mg%(R)	B/L Cataract
261.	Teerath Nath	49/M	130/70	None	NAD	Not done	Not done
262.	Baba	24/M	120/74	None	NAD	Not done	Not done
263.	Kalloo	33/M	116/70	None	NAD	Not done	Not done
264.	Sagarika Das	23/F	100/70	Burning micturition	Pus cells 16-20/HPF	88mg% (2hrpp)	Not done
265.	T. Mahapatra	25/M	110/50	None	NAD	Not done	Not done
266.	Amrit Singh	34/M		None	NAD	Not done	Not done
267.	Leena Devi	40/f	105/70	None	NAD	Not done	Not done
268.	PrabhuDayal	45/M	130/86	None	NAD	Not done	Not done
269.	Gayatri Devi	62/F	140/50	Known diabetes on drugs	NAD	210mg%(2hrpp)	B/L Cataract
270.	Eeshwar Das	59/M	170/90	None	NAD	Bl. Sugar=84mg% (R)	B/L(N)
271.	Ram Khilawan	41/M	110/70	Occasional renal angle pain	Pus cells 8-10/hpf Oxalate crystals +	Not done	Not done
272.	Krishna Sengar	32/F	100/70	None	NAD	Not done	Not done
273.	Gautam Jain	47/M	110/70	None	NAD	Not done	Not done
274.	Govind Singh	54/M	120/70	None	NAD	94mg% (2hrpp)	(N)
275.	Meena	5/F	110/70	None	NAD	75g%(4hrpp)	B/L (N)
276.	Veena	26/F	104/70	None	NAD	Not done	Not done
277	Katori Devi	33/F	1400/68	None	12-15/HPF Pus cells	Bl. Sugar=72mg%	Not done
278.	Kalyan	35/M	120/78	None	NAD	Not done	Not done
279.	Gaman singh	24/M	110/70	None	NAD	Not done	Not done

PART II –FOLLOW UP OF PATIENTS WITH ASYMPTOMATIC URINARY ABNORMALITIES

S.No	Name	Age/ Sex	Blood Pressure in mmHg	Symptoms	S. Creatinine	Repeat Urine (R,M)	USG for KUB	Biopsy/ Culture	Blood Glucose Fasting	Diagnosis
1	Shanti Bai	55/F	140/96	H/O swelling of whole body since last 2 years and abdominal pain 1 month	6.7	Protein++	B/L Shrunken kidney	Not done	94mg%	Symptomatic CRF
2	Smt. Sushma shivhara	32/F	120/72	Burning micturition last 3 days	0.9	10-12 pus cells\HPF	Normal kidney size with normal corticome dullary images I	E.coli in culture	77mg%	UTI
3.	Ram lal	64/M	130/90	Asymptomatic	0.7	Sugar++ puscells-6-8/HPF	NAD	Not done	321mg%	Diabetes Mellitus with U.T.I.
4.	Anjana	36/F	120/80	Asymptomatic	0.8	Sugar+++ puscells14-16/HPF	NAD	Not done	194mg%	Diabetes Mellitus
5.	Mohd Mustaq	51/M	150/98	Polydipsia polyurea	1.1	Sugar+++ puscells 4-6/HPF	NAD	Not done	248mg%	Diabetes Mellitus
6.	Kirti	10/F	110/74	Asymptomatic	1.2	Protein++	B/L Increased Cortical Echogenicity	Biopsy shows Focal segmental glomerulosclerosis	98mg%	focal segmental glomerulosclerosis
7.	L.K. Sharma	46/M	124/80	Asymptomatic	1.1	Puscells 10-15/HPFOxalate crystals++Phosphats+	B/L Renal Calculus	Not done		B/L nephrolithiasis
8.	Bhagwati	40/F	140/94	Arthralgia+insomnia	1.0	10-12 Pus cells/HPF	NAD	Not done	234mg%	Diabetes Mellitus
9.	Hadid khan	65/M	130/84	Asymptomatic	1.4	Protein++ Puscells6-8/HPF	B/Lmild Hydronephrosis with grade II BPH	Not done	50mg%	B.p.H. with U.T.I.
10-	Mohd Mustaq	51/M	120/82	Pain at the Tip of penis during micturition	0.5	Field full of pus cells	25mmrenal calculus in the renal pelvis	Not done	84mg%	nephrolithiasis
11.	Raj Kumari	35/F	130/84	Known C/O type II DM (M drugs)	1.0	Protein+	NAD	Note done	165mg%	Known Diabetic
12.	Noorjahan	55/F	120/76	Micturition	0.7	Oxalate Crystals ++	B/L multiple renal calculli	Not done	90mg%	B/L nephrolithiasis
13.	Akhtari bibi	45/F	130/80	Asymptomatic	1.0	Sugar++	NAD	Not done	245mg%	Diabetes Mellitus

14.	Chand bibi	50/F	120/70	Asymptomatic	1.19	Pus cells 8-10/HPF	NAD		94mg% Culture Shows E. coli growth	U.T.I.
15.	Avinash	8/M	110/70	Asymptomatic	1.14	Pus cells6-8/HPF	NAD	Voiding pyelogram shows reflux	100mg%	Vescico ureteral reflux
16	Chandra singh	45/M	124/80	Asymptomatic	0.9	Protein+	NAD	Not done	225mg%	Diabetes Mellitus
17.	Sukhiya	35F	110/74	Asymptomatic	0.97	8-10Pus cells/HPF	NAD	E.coli growth in culture	80mg%	U.T.I.
18.	Kamran	26/M	110/70	Asymptomatic	0.84	Abundant calcium oxalate cmprtal1-2 pus cells/HPF	7.5mmCalculus in lt Kidney	Not done	112mg%	Nephrolithiasis
19.	Gajendra	60/M	174/110	Asymptomatic	1.1	Protein++	NAD	Biopsy shows Bening Nephrotic disease	104mg%	Hypertensive Nephropathy
20.	Laxmi Shanker	41/M	200/120	Asymptomatic	0.74	Protein+	NAD	Benign Nephrosclorosis	95mg%	Hypertensive Nephropathy
21.	Ramkunvar	38/M	120/74	Asymptomatic	0.91	Pus cells16-18/HPF oxalatecryted plsent	B/L Renal Calculus	Not done	174mg%	Diabetes Mellitus
22.	Ramkumar	35/M	146/98	Cough+Lold	0.72	Sugar++occasioned pus cells	NAD	Not done	174mg%	Diabetes Mellitus
23.	Ram Avtaar	26/M	150/106	Occasional headache	0.97	Potein++	Grade II Renal Parenchyn el disease	Bipsy showsCGN	96mg%	CGN
24.	Nareshprasad	64/M	140/100	Asymptomatic	1.0	Pus cells8-10/HPF	Grade I BPH	Not done	104mg%	B.P.H
25.	Kasim Iqbaal	35/M	120/80	Asymptomatic	1.1	Pus cells 6-8/HPF RBCs 5-7/HPF Oxalate + phosphate crytals	Renal calculus Rt Kidney	Not done	74mg%	Nephrolithiasis
26.	Peelu Bhagat	40/M	126/74	Obesity	0.7	Sugar++ Protein+	NAD	Not done	251mg%	Diabetes Mellitus
27.	Shobha Das	15/F	110/74	Occasional buriney micturition	0.8	10-12 Pus cells /HPF	NAD	Culture shows E coli	100mg%	U.T.I.
28.	Tayanum Khan	30/F	100/70	Asymptomatic	0.78	Pus cells 10-12/HPF	Normal foctus of 26 week gst age KUB Normal	Not done	110mg%	U.T.I.
29.	Nidhi Pathak	10/F	110/90	Burning micturition	0.79	Pus cells10-14/HPF	NAD	Culture shows klebsiella	84mg%	U.T.I.
30.	Rambha Chaudhary	45/F	204/120	Asymptomatic	0.67	Protien++	NAD	Biopsy shows benign nephro sclerosis	110mg%	Hypertensive nephropathy

31	Neetu	8/F	150/90	Asymptomatic	1.12	Protein ++ RBC +	NAD		84mg%	IGA Nephropathy
32.	Alatash Khan	40/M	148/150	Asymptomstic	0.87	Sugar ++ Protein + Puscells 8-10/HPF	NAD	Not done	221mg%	Diabetic Mellitus
33.	Prakash Shukla	5/F	1401/100	Asymptomatic	1.8	Protein++	B/L renal parenchyn al disease Rt kidney size =6.57 x 4.3 mmRt kidney size normal	Not done	73mg%	Nephrolithiasis
34	Arjun Singh	20/M	124/76	Asymptomatic	0.8	Pus Cells 10-15/HPP	NAD	Streptococci growth		U.T.I.
35	B. K. Rai	35/M	160/116	Asymptomatic	0.94	Protein ++	NAD	Biopsy shows Benign Nephrosis	80mg%	Hypertensive Nephropathy
36.	Umar Mukhtar	42/M	150/100 mmHg	Asymptomatic	0.83	Pus cells10-12 /HPF	NAD	Not done	270mg%	Diabetes Mellitus with U.T.I.
37.	Pyaare mohan	32/M	124/76	Asymptomatic	01.71	Oxalate +phorpha te crystals	27 mmRt renal calculus	Not done	120mg%	Nephrolithiasis
38.	Kuhar Singh	24/M	200/100	Asymptomatic	0.98	Protein +	USG shows increased echogenici ty	Biopsy shows feature of CGN	80 mg%	Chronic GlomeruloNephriti s
39.	Lagan gupta	58/M	140/86	Poor steam formation during micturition	0.92	Pus cells 16-18/HPF	Grade II BPH others-NAD	Not done	104mg%	B.P.H.
40	Peelu singh	44/M	128/80	Asymptomatic	0.94	Protein++	B/L grade II renal parenchy mal disease	Biopsy shows Amyloid Deposition	88mg%	Renal Amyloidosis
41.	Man deep chaddha	45/M	160/104	Burning micturition	1.2	Pus cells full protein ++	B/L Increased Echogenici ty	Not done	294mg%	Diabetes Mellitus
42.	Preetam Srivasta va	22/M	198/120	Asymptomatic	1.02	Protein++	B/L renal parenchy mal disease (gradeII)	Biopsy shows CGN	93mg%	Chronic Glomerulo Nephritis
43.	Deewan Seth	58/M	160/100	Asymptomatic	1.8	Protein + Pus cells 6-8/HPF	B/L Renal parenchy mal disease grade I + BPH grade II	Biopsy shows Benign glomerulo sclerosis	81mg%	BPH with Hypertensive Nephropathy
44.	Prem Das	70/M	148/98	Know diabetes drugs	1.0	Protein ++	NAD	Not done	174mg%	Diabetes Mellitus

45.	Bheem Sen	34/M	120/80	Asymptomatic	0.94	crystas oxalate enypets	NAD	Biopsy Normal, serum calcium 16mg%.	94mg%	hyperparathyroidism
46.	Madhuri Gupta	8/F	110/70	Asymptomatic	0.84	Sugar ++	NAD	Not done	108mg	fanconi's syndrome
47.	Urmila Sethi	44/F	130/50	Asymptomatic	0.73	Sugar +++	8mm size calculus in the Rt lower ureter with 5mm calculus in the Rt upper caly	Not done	204mg%	Diabetes Mellitus with Renal Calculus disease
48.	Pardhan kumar	74/M	174/110	Asymptomatic	0.81	Protein++	Grate II renal yarnecbyal disease	Biopsy shows benign Nephro sclerosis	93mg%	Hypertensive Renal Disease
49.	Susheela Devi	50/F	100/70	Asymptomatic	0.79	Puscells1 4-16/HPF	NAD	Culture shows E. cells	100mg%	U.T.I.
50.	Kamini Begum	35/F	150/100	Poor vision	1.20	Protein ++	NAD	Not done	249mg %	Diabetes Mellitus
51.	Ramayya Devi	30/F	104/70	Burning micturition	0.95	Pus cells 14-16/HPF With traces of Albumin	NAD	Culture chows E-coli	89mg%	U.T. I
52.	Vishwanath Das	54/M	140/100	Asymptomatic	0.8	Protein+	USG shows grade II BPH	Not done	77mg%	BPH
53.	Meera Devi	65/F	120/50	Asymptomatic	1.24	Sugar +++ Protein +	NAD	Not done	344mg%	Diabetes Mellitus
54.	Sajid hussain	9/M	160/104	Puffiness of face & around eyes	1.64	Protein ++	B/L Grade II Renal parenchymal disease	Biopsy shows C.G. N.	94 mg%	C.G.N.
55.	Ladhun war	65/F	110/70	Burning during micturitum + proloper of uterim	1.12	Pus cells 10-12/HPF Traces of Albumin	NAD	Urine culture shows klebsiella	74mg%	U.T.I. with prolapse uterus
56.	Maan kunwar	58/F	210/120	Asymptomatic	1.69	Protein ++	NAD	Biopsy show Benign nephrosclerosis	70mg%	Hypertensive Nephropathy
57.	Pyaari	24/F	100/70	Asymptomatic	0.77	Pus cells 6-10/HPF	NAD	Not done	82mg%	U.T.I.
58.	Ganjani Devi	42/M	110/76	Asymptomatic	0.72	Pus cells 16-18/HPF	Normal	Not done	170mg%	DiabeteS Mellitus
59.	Malkhan singh	30/M	120/50	Asymptomatic	0.91	12-14 Pus cells/HPF	B/L renal calculus in the upper calyces(Rt. 8mm;Rt=12mm)	Not done	75mg%	Nephrolithiasis

60.	Premvat i	32/F	110/70	B/L occ. Renal angle pain	0.84	Pus cells 10-12/HPF few phosphat e cryplidd with oxalate cryrtas	B/L renal calcular disease (Rt 24mm cacular at low calyers) (Rt 10 mm calcular ain the Pelvo)	Not done	72mg%	Nephrolithiasis
61.	Rooma singh	16/F	110/72	Asymptomatic	0.77	14-16 Pus cells/HPF	NAD	Culture shows E-coli	94mg%	U.T.I.
62.	Om Dev	59/M	150/94	Known diabetes on drugs	1.0	Protein ++	NAD	Not done	200mg%	Diabetes Mellitus
63.	Allen Dsouza	35/M	188/104	asymptomatic	2.0	Protein + RBCs 6-8/HF	NAD	Biopsy shows Benign nephso	92mg%	Hypertensive Renal disease
64.	Barkat	45/M	110/80	asymptomatic	0.72	Pus cells 10-12/HPF	NAD	Culture shows klebsiells	104mg%	U.T.I.
65.	Rahman wllak	50/M	160/100	Asymptomatic	0.92	8-10Pus cells /HPF RBC 6-8/HPF	Grate II BPH	Biopsy – Normal	89mg%	BPH
66.	Saganik a Das	23/F	110/70	Burning micturituion	0.83	Pus cells 6-8/HPF	NAD	E.Coli in culture	50mg%	UTI
67.	Ram khilawa n	41/M	116/70	Rt Renal angle pain(occasimal)	1.20	Pus cells 8-10/HPF Occasinal oxalate crystals seen	A small 8mm calculus seen at the Rt lower calyx of the Rt gidney	Not done	74mg%	Nephrolithiasis
68.	Katori Devi	33/F	100/70	Asymptomatic	0.7	16-18 Pus cells /HPF	normal	E.Coli in culture	72mg%	UTI